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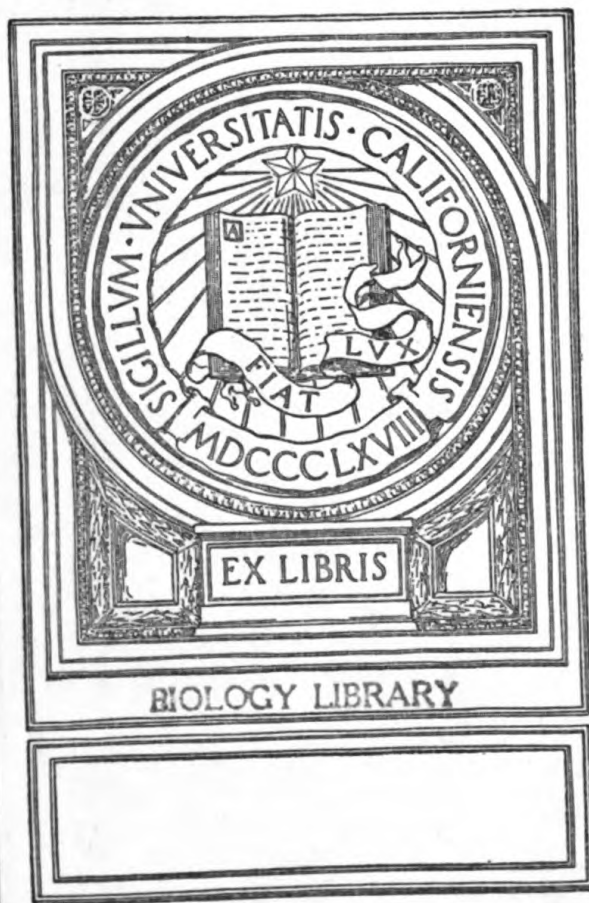


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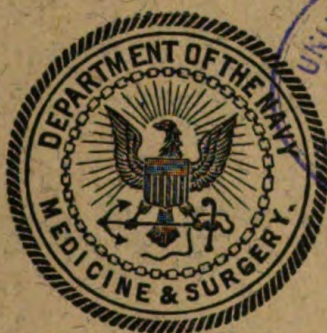
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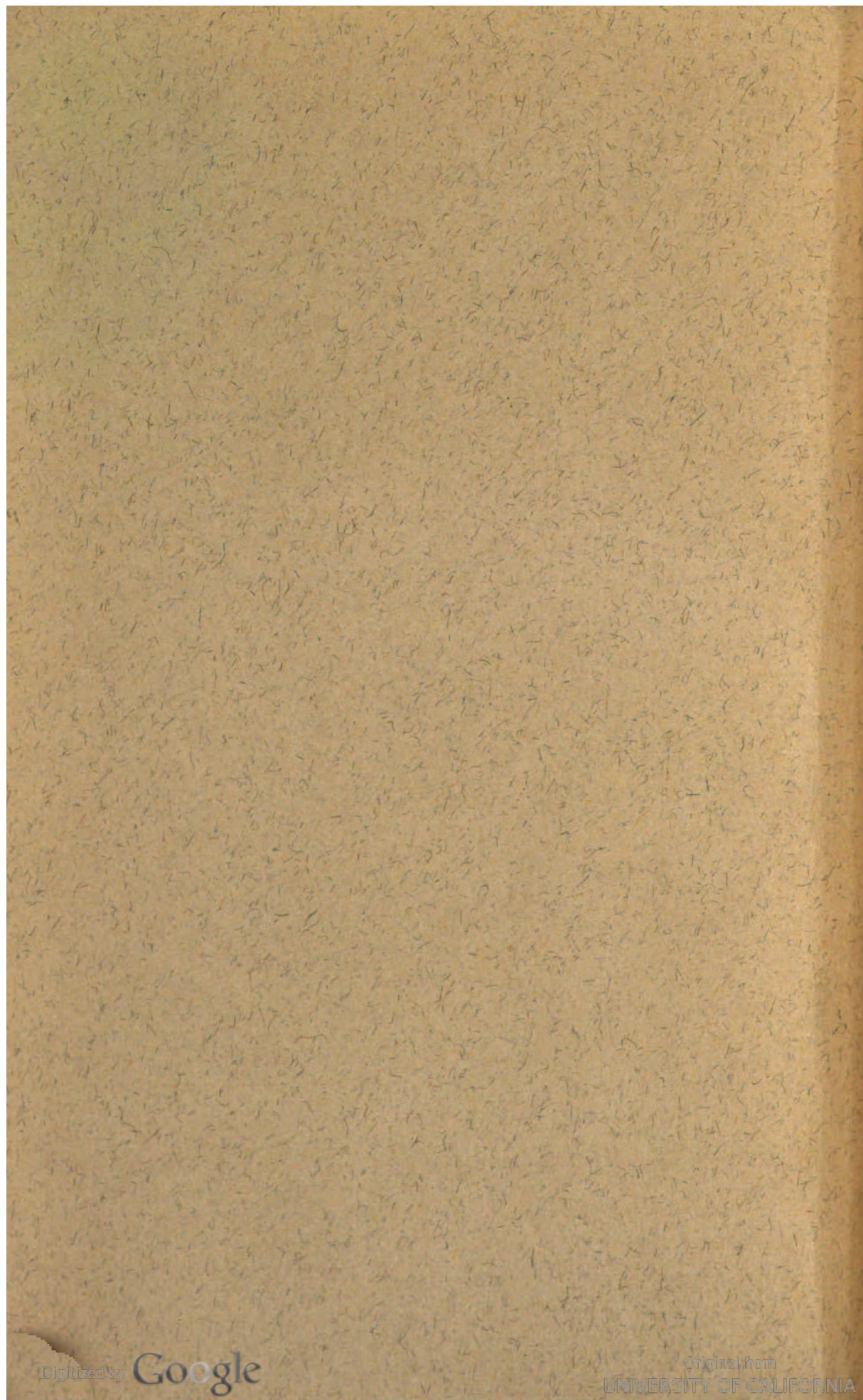
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JANUARY 1941

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UNITED STATES NAVAL MEDICAL BULLETIN

FOR THE INFORMATION OF
THE MEDICAL DEPARTMENT OF THE NAVY



DIVISION OF PUBLICATIONS
THE BUREAU OF MEDICINE AND SURGERY



THE MISSION OF THE MEDICAL DEPARTMENT OF THE NAVY



TO KEEP AS MANY MEN AT AS MANY GUNS
AS MANY DAYS AS POSSIBLE



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NAVY DEPARTMENT,
Washington, March 20, 1907.

This UNITED STATES NAVAL MEDICAL BULLETIN is published by direction of the Department for the timely information of the Medical and Hospital Corps of the Navy.

TRUMAN H. NEWBERRY,
Acting Secretary.

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PREFACE

THE UNITED STATES NAVAL MEDICAL BULLETIN was first issued in April 1907 as a means for supplying medical officers of the United States Navy with information regarding the advances which are continually being made in the medical sciences, and as a medium for the publication of accounts of special researches, observations, or experiences of individual medical officers.

It is the aim of the Bureau of Medicine and Surgery to furnish in each issue special articles relating to naval medicine, descriptions of suggested devices, clinical notes on interesting cases, editorial comment on current medical literature of special professional interest to Medical Department personnel, and reports from various sources, notes, and comments on topics of professional interest.

The Bureau extends an invitation to all medical and dental officers to prepare and forward, with a view to publication, contributions on subjects of professional interest.

In order that each service contributor may receive due credit for his efforts in preparing matter for the BULLETIN of distinct originality and special merit, the Surgeon General of the Navy will send a letter of appreciation to authors of papers of outstanding merit.

The Bureau does not necessarily undertake to endorse views or opinions which may be expressed in the pages of this publication.

ROSS T. MCINTIRE,
Surgeon General, United States Navy.

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Contributions to the BULLETIN should be typewritten, double spaced, on plain paper, and should have wide margins. Fasteners which will not tear the paper when removed should be used. Nothing should be written in the manuscript which is not intended for publication. For example, addresses, dates, etc., not a part of the article, require deletion by the editor. The BULLETIN endeavors to follow a uniform style in heading and captions, and the editor can be spared much time and trouble, and unnecessary changes in manuscript can be obviated if authors will follow in these particulars the practice of recent issues.

The greatest accuracy and fullness should be employed in all citations, as it has sometimes been necessary to decline articles otherwise desirable because it was impossible for the editor to understand or verify references, quotations, etc. The frequency of gross errors in orthography in many contributions is conclusive evidence that authors often fail to read over their manuscripts after they have been typewritten.

Contributions must be received at least 3 months prior to the date of the issue for which they are intended.

The editor is not responsible for the safe return of manuscripts and pictures. All materials supplied for illustrations, if not original, should be accompanied by reference to the source and a statement as to whether or not reproduction has been authorized.

The BULLETIN intends to print only original articles, translations, in whole or in part, reviews, and reports and notices of Government or departmental activities, official announcements, etc. All original contributions are accepted on the assumption that they have not appeared previously and are not to be reprinted elsewhere without an understanding to that effect and that editorial privilege is granted to this Bureau in preparing all material submitted for publication.

EBEN E. SMITH, *Editor*,
Commander, Medical Corps, United States Navy.

U. S. NAVAL MEDICAL BULLETIN

VOL. XXXIX

JANUARY 1941

No. 1

SPECIAL ARTICLES

SURGICAL HIGH LIGHTS OF 1940¹

By Commander F. R. Hook, Medical Corps, United States Navy

For the past several years Capt. L. W. Johnson of the Naval Medical Corps has prepared a yearly review of the surgical literature for the BULLETIN. These articles have been of great interest to readers of the BULLETIN and have had much to do in keeping those not in touch with a good medical library abreast of the times. Captain Johnson not only has a wide experience as a general surgeon but also possesses many other qualities that particularly fit him for such work, and it is unfortunate that his present duties have made it impossible for him to continue with these reviews. In substituting for him I have found the large amount of reading necessary, a pleasant and profitable task, but to know just what to retain and what to reject has not always been easy. Every naval medical officer should be fairly well acquainted with traumatic surgery; this is especially so at the present time, and it is for this reason that a great part of this work will consist of a review of the present trends in traumatic surgery.

WAR WOUNDS.—Statistics show that approximately 70 percent of wounds received during World War I were above the waist, trench warfare, naturally, being the cause of this. With the change from fixed trench to open warfare, and to the marked increase in bombing of civilians, the majority of wounds are now being reported as occurring below the waist.

Two new methods of wound treatment were developed during World War I. Carrel and Dakin were responsible for the continuous irrigation treatment with hypochlorite solution and secondary suture after the wound had become sterilized. Pierre Laval and others working in Flanders developed débridement. Both of these methods are based on the premise that all war wounds are infected. Both have their advantages and disadvantages. The success of débridement naturally depends to a great extent upon the thoroughness with which débridement is done but to a greater extent upon the time

¹ Submitted for publication October 28, 1940.

that has elapsed before treatment is instituted, for if infection has spread to the surrounding tissues the method is doomed to failure. Whitby and Camb² have shown that with the exception of violent and fulminating infection, there is ordinarily a lag period of 5 to 12 hours between the time of wounding and the time when the implanted infection becomes a dominating and often uncontrollable factor; this lag, they think, can doubtless be increased by prophylactic chemotherapy. Orr, based upon his experience in war surgery early in the twenties, developed a method of saucerization, packing the wound open with vaseline gauze, and encasing the extremity in a plaster cast. This method of treatment received quite wide usage in chronic osteomyelitis in civil practice but many traumatic surgeons were afraid to use it in acute traumatic lesions for fear of the plaster dressing obscuring a gas bacillus infection. Trueta,³ working in the recent Spanish Civil War, took up the Orr treatment, modified it, and has reported outstanding results with its use in 20,000 war wounds. The number of seriously infected wounds and gas gangrene following this method of treatment has been surprisingly small. Girdlestone⁴ and other English writers praise the method highly and say that it had much to do with the British wounded being returned from France in good condition. Trueta says the value of this form of treatment lies in:

1. Rest allows venous and capillary thrombi to form. These prevent and delay the spread of infection and are not broken down by repeated handling.
2. Rest allows new capillaries to form which are not torn by dressing of the wound.
3. The plaster maintains a constant beneficial pressure on the wound.
4. The mixture of organisms on the wound may by their mutual antagonism prevent the victory of any one group.

The fundamental requirement for both wound healing and for protection against infection is a good blood supply; lacerated, devitalized tissue, blood clots and foreign material encourage both aerobic and anaerobic infection. In doing débridement all tissue likely to become necrotic should be removed at the earliest possible opportunity. There should be no unnecessary sacrifice of undamaged tissue, and important nerve and blood vessel structures should be preserved whenever possible. If the wound is to be closed there should be no tension on the sutures. If the wound is to be left open the drainage should be free from all parts of the wound. The plaster should be put on properly by one who knows how. It should be nonpadded except over the bony prominences and should fit snugly and smoothly. It is impossible to lay down a hard and fast rule as to which wounds can be closed and which should be left open. Those favoring closure usually put 8 hours as the outside limit of safety for closure but prefer

² Whitby, L. E. H., and Camb, M. D., *Lancet* **238**: 655, April 16, 1940.

³ Trueta, J., Paul B. Hoeber Pub. Co., New York, 1940.

⁴ Girdlestone, G. R., *Lancet* **239**: 31, July 13, 1940.

to do it under 6 hours. Eloesser,⁵ who organized, staffed, and ran a hospital unit for 8 months on the Spanish front, is of the opinion that war wounds should practically never be closed unless the patient is to be kept under the observation of the surgeon doing the work.

Prophylactic serum against tetanus and gas gangrene should be given as soon as possible after the wounding. Even though many have their doubts as to the efficacy of antigas gangrene serum, they all agree that it should be used in the active treatment of the disease. Sewell⁶ says that with the use of irradiation, serum, and sulfanilimide the indications for amputation will be much less frequently found than 10 years ago. As a whole antiseptics have little place in the treatment of traumatic lesions. Rest, elevation, complete immobilization and adequate drainage are the things that count.

COMPOUND FRACTURES.—Sherman⁷ is not in accord with the methods being advocated in Europe of nonpadded plaster, closed technic of treating compound fractures. He states that such methods may be necessary in war surgery but in civil life where time permits accurate reduction of the fracture, immobilization with plates or by splints is the best procedure and will bring about the best results in the shortest period of time, with a minimum of deaths, sepsis, and amputations. Hermann⁸ thinks one should not be dogmatic about "closed and open" wounds and that the surgeon should be ready to use the one suitable for the case. He is also in favor of plating compound fractures and says that punctured wounds should be débrided just the same as lacerated wounds. Darrach⁹ is of the opinion that shock should not delay operative procedures beyond the time required for obtaining the necessary roentgenograms and preparation of the operating room unless the patient is pulseless or moribund. All are agreed upon the earliest possible definitive treatment with complete excision of all devitalized tissue and foreign material, the best possible alignment of fragments, and complete immobilization and rest.

Watson-Jones¹⁰ comes to the defense of the guillotine operation which Ogilvie has condemned by saying "the guillotine amputation is a crime which no conceivable surgical or military experience can justify." Watson-Jones says that if properly done it has a place in war surgery and complete success demands 3 conditions be observed: (a) The amputation must be at the lowest level of the limb, the division of the bone being in a plane slightly above rather than slightly below that of the soft tissues, (b) skin traction to a Thomas splint or to weights over the foot of the bed must be applied at once

⁵ Eloesser, Leo, Clin. Prof. Surg., Stanford Univ., Personal Communication.

⁶ Sewell, R. L., Surgery 6: 221, August 1939.

⁷ Sherman, W. O., Arch. Surg., 40: 838, May 1940.

⁸ Hermann, O. J., Arch. Surg., 40: 853, May 1940.

⁹ Darrach, Wm., Arch. Surg., 40: 821, May 1940.

¹⁰ Watson-Jones, R., Lancet 239: 186, August 10, 1940.

and not after several days or weeks, (c) the vaseline gauze "no dressing" technic should be employed.

SHOCK.—There probably is no subject in medicine that has received more study through the years than that of shock, and still its cause in many instances is unknown. Cressman and Blalock ¹¹ state that the most fundamental abnormality in shock is disproportion in the size of the vascular system and its contents. This may be due to a marked increase in the size of the vascular system or decrease in the blood volume, or both, which results in systemic anoxia. Moon ¹² says that shock may originate from numerous and varied agents and conditions which may injure directly the capillary endothelium, from loss of blood or fluid or both sufficient to produce systemic anoxia, from agents or conditions which reduce the volume flow of blood below physiologic limits, from asphyxia of external or internal origin, or from various combinations of the conditions mentioned. Shock unaccompanied by significant hemorrhage is characterized by hemoconcentration, the degree of which is in proportion to the degree of shock. All authorities are agreed that the advances in prevention of shock have gone ahead of the methods of treating fully developed shock, and until some agent is found which will prevent or relieve capillary atony, efforts to interrupt the cycle should be directed toward the restoration of blood volume and toward relieving the anoxia. Of importance in the prophylaxis of shock as outlined by Blalock ¹³ are:

The condition of men as to food, fluids and nutrition, first-aid dressings, and subsequent care early in the treatment of wounds, control of hemorrhage, relief of pain, immobilization of fractures, prevention of chilling especially if clothing is wet, vaso-constrictor drugs (of little use), administration of fluids intravenously and by hypodermoclysis, and the inhalation of oxygen.

In the treatment of well-developed shock the intravenous administration of isotonic solution of crystalloids has at most only a temporary beneficial effect as the fluid soon passes from the capillaries into the tissues. At the present time blood plasma is the most valuable, effective, and practical method in the prevention and treatment of shock. Whole blood may, however, be preferable where marked hemorrhage has occurred. Selge and Dosne ¹⁴ have recently reported upon their experimental work on the rat which showed that pure corticosterone administered in an aqueous solution is very effective in combating shock caused by surgical trauma.

TRANSFUSION.—Many lives have been saved by the institution of blood banks in the various medical centers throughout the country. Now that they have been in existence for some time considerable

¹¹ Cressman, R. D. and Blalock, A., *Amer. Jour. of Surg.*, **46**: 417, December 1939.

¹² Moon, V. H., *Jour. Amer. Med. Assn.*, **114**: 1312, April 6, 1940.

¹³ Blalock, A., *Manual on Shock*, Natl. Def. Council.

¹⁴ Selge, Hans, and Dosne, C., *Lancet* **239**: 70, July 20, 1940.

information on stored blood has accumulated. DeGowin and Hardin ¹⁶ state that the incidence of all types of reaction from transfusion with preserved blood is no greater than with fresh blood providing proper care is taken in storing and handling of it, and that the time limit of storage is 10 days for citrated blood and 30 days for dextrose-citrate mixture. Hemolysis is retarded when large amounts of glucose are added to the blood but the dilution factor is objectionable as Blalock ¹³ has pointed out. Owing to migration of the potassium ions from the cells into the plasma he feels that stored blood more than 8 days old should not be used. Edwards and Davie ¹⁶ also advise against using blood more than 10 days old.

Although blood banks function well in our cities and medical centers many objections are raised against them in the treatment of war wounds away from these centers. A large, expensive, cumbersome unit is necessary to care for the stored blood, compatibility tests are necessary, the length of time that blood can be stored with safety is limited, and the agitation associated with transportation increases the rate of hemolysis and decreases the safety time of the blood. Blalock ¹³ states that those wounded will stand a tremendous decrease in the volume of red blood cells providing the plasma volume is not markedly reduced, and that most patients who die from loss of red blood cells do so before medical aid is available. Loss of red blood cells is tolerated well if the plasma loss is replaced. For this reason blood plasma or serum has the following advantages over whole blood: It can be stored for months without significant alteration, it can be transported with less difficulty, it can be used without reactions and without laboratory typing, it is ready for immediate use, and it does not add to the concentration of the red blood cells. Levinson, Neuwelt, and Necheles ¹⁷ and Elliott, Busby, and Tatum ¹⁸ report favorably upon the effectiveness of plasma as a substitute for whole blood and to the rarity of reactions from its use. Plasma may be stored in the liquid form or it may be dried and sealed in a tube under vacuum and will keep for at least a year. Approximately 8 grams of the dried product are obtained from 100 cc. of citrated blood. The plasma may be regenerated simply by adding sterile distilled water previous to its use. Several methods of drying plasma are now available but all have the disadvantage of being slow and costly. The need for a dried plasma is not so important in the navy as it is in the army as the fluid plasma properly prepared will keep indefinitely, it can be stored aboard ship without difficulty, and would be available for immediate use.

¹⁶ DeGowin, E. L. and Hardin, R. C., *Jour. Amer. Med. Assn.*, 115: 895, September 14, 1940.

¹⁶ Edwards, F. R. and Davie, T. B., *Brit. Med. Journal*, 2: 73, July 20, 1940.

¹⁷ Levinson, S. O., Neuwelt, F., and Necheles, H., *Jour. Amer. Med. Assn.*, 114: 455, February 10, 1940.

¹⁸ Elliott, J., Busby, G. F., and Tatum, W. L., *Jour. Amer. Med. Assn.*, 115: 1006, September 21, 1940.

TETANUS.—Several excellent articles have appeared in the past year on this disease. Moore and Singleton¹⁹ state that in the past 15 years the death rate has been reduced by one-half and that in the United States there still are 1,000 deaths a year from tetanus. Deaths are more common in the southern states in the Negro population. This is thought to be due to economic and occupational influences rather than racial susceptibility. Dietrich, Karshner, and Stewart²⁰ reporting on tetanus treated at the Children's Hospital, Los Angeles, state that during the past 18 years the mortality rate has dropped from 80 percent to 8 percent. Of the patients surviving, 69 percent showed lesions (compression of one or more bodies) in the thoracic spine. During the period 1921 to 1933, fifteen patients were treated at that hospital, 12 of whom died. The treatment during that period consisted of débridement and the injection of antitoxin in large amounts, locally, intramuscularly, intravenously, and intrathecally. Very little sedation was given. The patients in this group received 15,000 to 100,000 units of antitoxin. Many of these patients appeared to be relatively mild cases but all had severe reactions after antitoxin was given and, in 3 to 14 hours, 11 of the 15 patients were dead. From 1933 to 1938, thirteen patients were treated with 1 death, this patient dying 3 hours after the first administration of 20,000 units of antitoxin intravenously. In only 4 patients in this group was antitoxin given intrathecally and all of these had severe reactions. They are very emphatic in saying that antitoxin should never be given intrathecally in juvenile tetanus because of the severe reactions which result in sterile meningitis, cerebral edema, and death. They feel that the use of antitoxin intravenously is safe provided that adrenalin is given in conjunction with the introduction of any serum in the vein. Relatively small doses of antitoxin are used and much dependence is placed on sedatives for they save lives and are valuable in preventing vertebral deformities. Treatment of the spinal lesions, once established, they say, is of no avail. Experimentally, Shumaker, Firor, and Lamont²¹ report that tetanus antitoxin administered intrathecally is superior to that given intravenously in guinea pigs and Firor²² has shown that intracisternal injections of antitoxin into dogs suffering from early, mild, or moderately severe tetanus yield far better results than intravenous injections.

Surgeons learned during the last war to repeat the prophylactic antitoxin in the severely wounded, especially if surgery was done at some later date, as it was found that antitoxin gave an immunization of only 4 to 11 days. Sufficient evidence is at hand now to say that

¹⁹ Moore, R. M., and Singleton, A. O., S. G. and O., 69: 146, August 1939.

²⁰ Dietrich, H. F., Karshner, R. G., and Stewart, S. F., Jour. Bone and Joint Surgery, 42: 43, January 1940.

²¹ Shumaker, H. B., Firor, W. M., and Lamont, A., Surgery, 8: 1, July 1940.

²² Firor, W. M., Arch. Surg., 41: 290, August 1940.

toxoid will give a relatively lasting immunity. Firor²³ says that all French soldiers and horses were given toxoid. In the past they lost 40 to 60 horses a year in this group but last year not a horse died among the 60,000 that had been actively immunized with toxoid. He recommends 3 injections of 1.0 cc. of the purified alum precipitate given at intervals of 6 to 12 weeks. After a patient has had these 3 injections a fourth will rapidly raise the titer of antitoxin in his serum to a level which is absolutely capable of protecting him from tetanus. This is so prompt that no antitoxin need be given. He summarizes the treatment of tetanus as follows: (a) An initial dose of 50,000 units of antitoxin intravenously, (b) infiltration by multiple injections of 10,000 units of antitoxin around the site of the injury, (c) an hour after this local injection, excision of the offending wound, (d) daily injections of 5,000 units of antitoxin, (e) sufficient fluids preferably by mouth or through the nasal tube to keep the patient in fluid balance, (f) adequate nourishment, (g) control of convulsive seizures by the use of paraldehyde, chloral or ether-oil, (h) the use of the respirator when necessary, and (i) tracheotomy for laryngeal spasm causing suffocation.

Bryant and Fairman²⁴ report 22 patients with tetanus treated with M&B 693 (sulfapyridine) and by continuous or intermittent narcosis with sodium evipan with only 5 deaths. Evipan is given to produce continuous drowsiness and the dose is increased if necessary until all convulsions have been controlled. Usually 1 gram night and morning is all that is necessary but at times an additional gram at midday may be required. Narcosis may be prolonged by morphine and other sedatives. The dosage of M&B 693 is given as 1 tablet dissolved in 5 to 10 cc. of normal saline and injected intramuscularly.

Tetanus has been reported following burns and frostbite. Eloesser²⁵ says that 10 percent of the patients with frozen feet in the Spanish Civil War developed tetanus.

BURNS.—Of the many interesting papers appearing on this subject during the past year, that of Elkington, Wolff, and Lee²⁵ on plasma transfusions in the treatment of the fluid shift in severe burns is deserving of special review. They had previously stressed the importance of the early local treatment of burns and this paper dealt chiefly with the maintenance of fluid balance. Their work has further substantiated the view that the fluid imbalance in severe burns is primarily due to an altered capillary permeability with a shift of fluid protein into the tissues, rather than an external loss, and that the restoration of plasma protein by means of plasma transfusions is a rational treatment for this fluid loss.

²³ Firor, W. M., *Amer. Jour. of Surg.*, 46: 450, December 1939.

²⁴ Bryant, J., and Fairman, H. D., *Lancet*, 239: 263, August 31, 1940.

²⁵ Elkington, J. R., Wolff, W. A., and Lee, W. E., *Ann. Surg.*, 112: 150, July 1940.

They say that the plasma protein loss continues for as long as 31 to 40 hours and that during this period excessive hemoconcentration can be prevented by repeated small plasma transfusions. After the fortieth hour when the capillaries have regained their impermeability to protein, the deficit of plasma protein may be corrected quantitatively by a large plasma transfusion. The amount of protein required is calculated by a formula based on hematocrit values, plasma protein concentration, and body weight. This regimen permits the restoration of plasma volume to normal without the administration of excessive amounts of protein-free fluids. In one of their cases they report a loss to 63 percent of the normal plasma volume within 40 minutes after the burn; there was a corresponding rapid loss of plasma protein. The administration of 6,000 cc. of protein-free fluids in the course of 24 hours did not affect the plasma volume but there was an immediate response to transfusion of 1,400 cc. of plasma protein. There was a loss of most of this administered plasma protein within 6 hours with a corresponding drop in the plasma volume. A plasma transfusion at 66 hours returned the plasma volume to approximately normal, where it remained.

Trusler, Egbert, and Williams²⁶ are of the opinion that the tannic acid theory of the treatment of burns is fallacious, that there is no local application that can save life after a large burn, and that the first cause of death is shock. They point out that the indiscriminate forcing of simple fluids into a burned patient is futile and may lead to profound disturbances in the physical-chemical relation of the blood. They recommend early and repeated blood transfusions and large amounts of dextrose throughout the self-limited period of reaction. In one of their patients following six large blood transfusions in 4 days blood plasma was substituted for whole blood due to polycythemia. Blackfield and Goldman²⁷ stressed the necessity of hospitalizing all children with severe burns due to the doubtful prognosis. They condemn the use of unguents and recommend the tannic acid bath and silver nitrate or gentian violet method of treatment. One might summarize the present treatment of burns as follows: (a) Early local treatment with tannic acid and silver nitrate; (b) early and repeated small plasma transfusions if available; if not, transfusions with whole blood; (c) administration of sulfanilimide or one of the other sulfonamides; and (d) skin grafting at the earliest possible time to prevent septic toxemia and contractures.

CONTROLLED FLUID THERAPY.—It should be remembered that a poorly managed pre- or post-operative regimen may vitiate the most brilliant surgery. Drew, Scudder, and Papps²⁸ state that four simple

²⁶ Trusler, H. M., Egbert, H. L., and Williams, H. S., *J. A. M. A.*, 113: 2207, December 16, 1939.

²⁷ Blackfield, H. M., and Goldman, L., *J. A. M. A.*, 112: 2235, June 3, 1939.

²⁸ Drew, C. H., Scudder, J., and Papps, J., *S. G. & O.*, 70: 859, May 1940.

tests may be used as emergency measures in determining and regulating the state of hydration in acutely ill persons. They are: (a) The determination of the cell volume of venous blood by the hematocrit, (b) determination of the specific gravity of the whole blood by the modified Balfour and Hamilton method, (c) determination of the specific gravity of the plasma, and (d) calculation of the plasma proteins from the plasma specific gravity by a simple formula. By means of the data thus acquired one may determine the degree of water loss, water plus protein loss, anticipate the onset of shock, differentiate shock due to simple circulatory collapse from shock complicated by hemorrhage, detect dehydration in the presence of anemia, predict the approach of an edema level of proteins, and direct treatment more rationally for the alleviation of any of these conditions. Latimer ²⁹ says a simple test of hydration is to measure the 24-hour urinary output which should be approximately 1,500 cc. In any case of serious dehydration the water loss amounts to 6 percent of the body weight. The average daily sodium chloride requirement of an adult is 4.5 grams. The average patient unable to take fluids by mouth should receive 500 to 1,000 cc. of physiologic salt solution and the balance of the water requirement should be given as 5 percent dextrose in distilled water.

PENETRATING STAB WOUNDS OF THE ABDOMEN.—Wright, Wilkinson, and Gaster ³⁰ point out the necessity for immediate surgical investigation of all stab wounds of the abdomen and abdominal wall. They say that spinal anesthesia should never be used with this type of surgery as it increases peristalsis and adds greatly to the spill of intestinal contents into the abdominal cavity. Hamilton ³¹ agrees that all such injuries should be explored even though a surprisingly large number of laparotomies are performed without finding any perforation. He suggests the use of the peritonscope and cites five cases successfully managed in this manner.

FRACTURE OF THE NECK OF THE FEMUR.—Evidence continues to accumulate showing the advantages of internal fixation in the treatment of fractures of the neck of the femur over the older type of plaster and bed treatment. Rankin ³² states that where union was previously reported in 38 to 65 percent (average 50.4 percent) with a mortality rate of 28.6 percent following plaster treatment, with the internal fixation method, union occurs in 75 to 96 percent with a greatly reduced mortality rate. From an economical standpoint the internal fixation method is much superior to the older plaster method as the stay in the hospital is much reduced and the return to normal function is much earlier. It appears to make little difference whose method of

²⁹ Latimer, E. O., *American Journal of Surgery*, 48: 223, November 1939.

³⁰ Wright, L. T., Wilkinson, R. S., and Gaster, J. L., *Surgery* 6: 241, 1939.

³¹ Hamilton, J. E., *Surgery*, 7: 582, April 1940.

³² Rankin, J. O., *Ann. Surg.*, 3: 315, February 1940.

internal fixation is used. Even in intertrochanteric fractures which we have been taught in the past heal without difficulty and deformity, Callahan ³³ now recommends nailing to prevent shortening of the extremity.

FRACTURE OF RIBS.—In the treatment of fractured ribs Rovenstine and Byrd ³⁴ say that the relief from pain is important for the patient's comfort but more so as a prophylactic against respiratory infections which are responsible for most of the deaths following these injuries. Large amounts of opiates are contraindicated as they interfere with adequate lung ventilation and cause stasis of secretions by depressing respirations and the cough reflex. They use paravertebral injections of procaine and alcohol at the site of the involved thoracic nerves.

Schnur ³⁵ found that by injecting 5 to 10 cc. of a 2-percent procaine solution, pain was relieved in 32 patients suffering with pleural pain, and in 21 of these the relief was permanent. With the relief of pain respiration was definitely altered, inspiration became deeper, the rate slowed, expiratory grunt ceased, several patients appeared to have less cyanosis, cough became less disturbing and restlessness and insomnia disappeared. The patients showed improvement in their mental attitude and many fell into sleep within 10 minutes after the injection.

CHEMOTHERAPY IN SURGERY.—The reports of Freeman ³⁶, of Meleney and Harvey, ³⁷ and of Shallow, Fry, and Pulaski ³⁸ all speak well for the results obtained by the use of zinc peroxide in the treatment of chronic undermining, burrowing ulcers due to the micro-aerophilic hemolytic streptococcus. They lay stress upon the importance of early diagnosis through recognition of the clinical manifestations and isolation of the organisms by securing anaerobic as well as aerobic cultures. They recommend that sulfanilamide be given in conjunction with the zinc peroxide treatment.

Lyth ³⁹ reports favorably upon the use of saturated solution of sodium sulfate in the treatment of infected wounds. Due to its osmotic properties it is superior to other salts. He has used it in 1,096 cases, all of which recovered with the exception of malignant ulcers.

Shinna ⁴⁰ recommends for infected wounds a solution of chlorine, iodine, picric acid, and zinc sulfate. He states that the picric acid and zinc sulfate coagulate and rid the wound of proteins by precipitation allowing the chlorine and free iodine to display their disinfectant action.

³³ Callahan, J. J., Prof. of Surg., Cook Co. Grad. School, Personal communication.

³⁴ Rovenstine, E. A., and Byrd, M. L., *Amer. Jour. of Surg.* **46**: 303, November 1939.

³⁵ Schnur, S., *Ann. of Int., Med.*, **13**: 845, November 1939.

³⁶ Freeman, B. S., *J. A. M. A.*, **115**: 181, July 20, 1940.

³⁷ Meleney, F. L., and Harvey, H. D., *Ann. Surg.* **110**: 1069, December 1939.

³⁸ Shallow, T. A., Fry, K. E., and Pulaski, E. J., *S. G. & O.*, **70**: 787, June 1940.

³⁹ Lyth, J. C., *Lancet* **238**: 216, February 3, 1940.

⁴⁰ Shinna, S., *Bul. of Nav. Med. Assn. (Japan)* **19**: 42, August 15, 1940.

The medical literature has contained much material on the sulfonamides in the past few years and a general review of the subject is published elsewhere in this issue. What interests us chiefly as naval surgeons at this time is the prophylactic use of these drugs in the treatment of the wounded. Reports from Europe are still meager but the English speak highly of the use of sulfanilamide, saying that it in conjunction with the Trueta treatment, had much to do with the wounded being returned from France in good condition. Reports are to the effect that they give an original dose of 6 to 7 grams of the drug immediately following the wounding and 1 gram every 4 hours thereafter throughout the 24 hours. There is a growing feeling in this country that the dangers of sulfanilamide have been oversold and that if the maximum benefits from its use are to be had large doses should be given. The committee on chemotherapy working under the medical section of the National Defense Council are now making studies along these lines and it is probable that a manual on the subject will be available for the use of the armed forces previous to the publication of this article.

Jensen, Johnsrud, and Nelson⁴¹ and others have reported good results from the instillation of crystalline sulfanilamide in compound fracture wounds. Some, however, think that there is no advantage in local application over that of taking the drug internally. They warn that the use of sulfanilamide in the wound is not a substitute for débridement.

The bacterial properties of penicillin, the active principle obtained from the mould *Penicillium notatum* has recently been reported upon by the Oxford School of Pathology⁴² and is of considerable interest as it has been demonstrated to be active both in *vivo* and in *vitro*, and therefore has claims as a chemotherapeutic agent. Preliminary reports state that the substance in a dilution of one in several hundred thousand inhibits the growth of such pathogens as gas gangrene anaerobes, streptococcus pyogenes, pneumococcus, and diphtheria bacillus. In mice experimentally infected with *Streptococcus pyogenes*, *Staphylococcus aureus*, and *Clostridium septique* treatment begun at the time of infection and continued at short intervals for 2 to 4 days in the case of the pyogenic cocci and 10 days for *Clostridium septique* saved most of the animals whereas all of the control animals died. No studies are available on the results of treatment of already established infection or upon the optimum therapeutic dose.

GASTRIC CANCER.—Schindler and Gold⁴³ state that gastric cancer in most cases develops upon the soil of chronic gastritis and that

⁴¹ Jensen, N. K., Johnsrud, L. W., and Nelson, M. C., *Surgery*, 6: 1, July 1939.

⁴² Chain, E., Florey, H. W., Gardner, A. D., Heatley, N. G., Jennings, M. A., Orr-Ewing, S., and Sanders, A. G., *Lancet* 239: 226, August 24, 1940.

⁴³ Schindler, R., and Gold, R. I., *S. G. & O.* 69: 1, July 1939.

atrophic gastritis is evidently a precancerous condition and should be diagnosed. Patients suffering from this condition should be examined by x-ray and gastroscope at regular intervals regardless of symptoms as a cancer producing symptoms is usually not of minimal size; this is particularly true in the body of the stomach. They also feel that patients with gastric ulcers who are more than 35 years of age should be examined by the gastroscope before starting medical treatment as the niche may become smaller during roentgen-ray observation, even though the lesion is malignant. The gastroscope has proved superior to the x-ray examination in determining the operability of certain cases; however, the roentgen-ray and gastroscopic examination should not be considered as competitive but each should supplement the other if the best results are to be had.

ANESTHESIA.—During the past year pentothal sodium has rapidly increased in popularity and threatens to replace the inhalation anesthetic agents for certain types of work. When properly given it apparently has a wide margin of safety. The ease with which anesthesia is induced and the absence of postoperative discomfort are particularly pleasing to the patients. This type of anesthesia should be ideal for war surgery at the front due to the minimal amount of equipment necessary for its use.

Lemmon⁴ has been able to overcome the chief disadvantage of spinal anesthesia by working out a method of giving continuous spinal anesthesia. By using a thick operating table pad with a cutout in it, he is able to leave the spinal needle in place and inject the anesthetic agent as needed. He uses procaine hydrochloride as it is less toxic than other agents. The level of analgesia is controlled by the position of the patient, dilution of the analgesic drug, and the force of the injection.

The subject of operating-room explosions received a great deal of attention at the convention of the American Hospital Association at Boston, September 1940. Capt. Lucius W. Johnson (MC), U. S. Navy, attended this meeting and in a personal communication to the author reports a particularly interesting demonstration, given by Dr. Horton, Professor of Biological Chemistry at the Massachusetts Institute of Technology. All aspects of these accidents were considered, and it is believed that definite progress has been made in eliminating them. Professor Horton showed how every move that we make in the operating room generates static electricity. When two objects having different potentials approach, there may be a discharge from one to the other sufficient to cause a spark which will ignite any explosive gases. He put his finger on an electrometer and scuffed his foot on the floor. The electrometer showed a sudden rise of 200 volts. He held a pillow while another man pulled the cover off it and the

⁴ Lemmon, W. T.: *Ann. Surg.* 3: 141, January 1940.

potential of 2,000 volts was developed. He scraped his arm with a piece of the material that nurses' uniforms are made of. It showed a rise of over 200 volts. These potentials are quite sufficient to give a large spark.

He described the intercoupler which connects the patient, the anesthetist, the operator, the anesthetic machine and the rubber bag containing the gases, and maintains them all at the same potentials so that there is no spark.

Then he talked on modern developments in conductive rubber. He substituted a strip of this conductive rubber for the ignition wire of a gasoline engine and kept it running. The same rubber can be used to make pads for the operating room table and stools, for floor material, for the rubber bags of anesthetic machines, for the soles of shoes; also for the casters of operating room tables and wheel stretchers. This prevents the accumulation of a high potential of any object. Standing on a sheet of conductive rubber, he repeated his experiments of scuffing his foot, pulling the cover off the pillow, and there was not enough rise in potential to be indicated on the electrometer.

The conductive rubber is produced by using acetylene black as a filler instead of the usual carbon black, and a high degree of conductivity can be obtained.

The large percentage of the acetylene black that is used precludes the possibility of having any other color than black. It also makes the rubber lose some of its elasticity. The DuPont Co. claims that with the use of their neoprene—an artificial rubber—there is no loss of elasticity.

He demonstrated the explosive power of various anesthetic gases, drawing them off in soap bubbles and exploding them by passing electric sparks through them. Then he demonstrated that there is a certain percentage of cyclopropane and ethylene mixture which is not explosive within the ranges used for anesthesia. By adding small amounts of helium or nitrogen, the explosiveness is further reduced.

Explosiveness of anesthetic gases mixed with oxygen

Anesthetic	Low limit	High limit	Most explosive
	<i>Percent</i>	<i>Percent</i>	<i>Percent</i>
Ether.....	2. 1	82	3. 5 to 7. 5
Ethylene.....	2. 9	80	27
Cyclopropane.....	2. 5	63	20

A mixture of ethylene 65 percent, cyclopropane 10 percent, and oxygen 25 percent is not explosive. Adding a little hydrogen increases the limits of the field of safety. This will cover the anesthetic range through from light to heavy anesthesia. The weak point in this is

that one never knows exactly the composition of gases within the rubber bag of the anesthetic apparatus.

High relative humidity offers a certain degree of protection against explosion, but this is fallacious because it is protective only if the carbon dioxide content is high. The Boston Woven Hose & Rubber Co. has developed a complete series of conductive rubber materials for operating-room use. Professor Horton regarded shoes as probably the most important factor in protection, and discussed the several methods of providing conduction from the body to the brass grill which is inlaid in the floor. He showed that many of the substances that are supposed to be conductive have a lag in the conduction, so that it may take one to two ten-thousandths of a second to discharge the body's potential, while it takes only one-millionth of a second to produce the spark that will cause a fatal explosion.

Floors will soon be available in conductive rubber or similar materials. These floors must never be waxed for the wax coating is an insulator.

It is recommended that terrazzo, marble tile, and other floors of this type after cleaning be flushed with a solution containing 2 to 4 percent of calcium chloride. This holds some moisture on the surface of the floor and makes it a better conductor. Conductive substances may be quite resistant and this is an important consideration in determining the safety of any appliance.

Explosion-proof operating-room lights have been produced by some makers and widely advertised. The underwriters in certain localities require that the operating-room lights be explosion proof.

Explosion proof means that the light is able to withstand an internal pressure of 500 pounds to the square inch. This means that the lens which transmits the light must be extremely thick. The metal shell must also be thick. This results in great loss of light and loss of adjustability because of the great weight of the fixture. So far as is known, no anesthetic-gas explosion has ever been due to an operating room light; and the experts say that what is required in the light is not that it be explosion proof but to be ignition proof so that no spark can be produced by its lights which will cause an explosion.

SUTURE MATERIAL.—There has been a definite trend the past few years away from absorbable (catgut) to nonabsorbable (silk, cotton, wire) sutures. Each type has its advantages and disadvantages and if these are kept in mind by the surgeon he will be able to choose the suture material most suitable for the case. The thing to remember is that many excellent surgeons are entirely satisfied with the use of absorbable sutures while others are equally satisfied with nonabsorbable suture, all of which leads one to believe that it is chiefly a matter of personal choice. If, however, one is to use silk he should conform

to the technic laid down by its advocates if the best results are to be obtained, and not use it in a careless, haphazard sort of way.

There is no question but that catgut of too large size has been used in the past and there is now a tendency to reduce not only the size but also the amount placed in wounds. Howes ⁴⁶ has shown by experimental work that catgut No. 0 is strong enough, as the holding-power strength of it is greater than that of the tissues, and the use of any larger material simply adds an excess of foreign material to be absorbed.

OPERATING ROOM.—In the Presbyterian Hospital, New York, Kraissl, Cimiotti, and Meleney ⁴⁶ report that by the use of ultraviolet radiation in the operating room they have reduced the number of infections from 4 percent serious and 10 percent trivial in 1925 to 0.5 percent serious and 1.6 percent trivial in 1938.

VITAMINS IN SURGERY.—Vitamin K has stood the test of time and trial so that now no surgeon would attempt to do surgery in jaundiced cases without first determining the prothrombin level and giving this vitamin if indicated. It has been the means of saving many lives. Pueston ⁴⁷ summarizes the use of vitamins in surgery by saying, "they play an important role in the state of general nutrition, cardiac reserve, resistance to infection, and in certain specific functions such as healing of wounds and the prevention of hemorrhage. In the preparation of surgical patients and in the postoperative care vitamin therapy must be accorded a place of prominence along with fluid balance and other nutritional considerations."

CARBUNCLES.—The swing from surgery to x-ray therapy in the treatment of carbuncles and furuncles the past few years has been slow but constant so that now many surgeons refuse to operate upon them. The relief from pain that patients with massive carbuncles frequently get from x-ray therapy is amazing. This therapy also shortens the period of illness and reduces the mortality rate. O'Brien ⁴⁸ reports on 130 cases of severe carbuncle treated in the Boston City Hospital with x-ray alone, or in conjunction with surgery; the mortality rate was 3 percent. Sixty patients were treated by x-ray alone, their hospital stay was the shortest of the entire group and only 1 died. There were no deaths in 57 cases of fascial carbuncle treated alone or chiefly by x-ray. Sulfanilamide has been of little aid in the treatment of carbuncles; sulfathiazole may prove to be more efficacious.

POLYPOID DISEASE OF THE COLON.—There is a growing belief among many authorities that most cancers of the colon arise from malignant degeneration of polypoid lesions. Hedin ⁴⁹ says that almost all recorded cases of multiple polyps of the colon eventually become

⁴⁶ Howes, E. L., *Surgery*, 7: 24, January 1940.

⁴⁶ Kraissl, C. J., Cimiotti, J. G. and Meleney, F. L., *Ann. Surg.* 3: 161, February 1940.

⁴⁷ Pueston, C. B., S. G. & O., 70: 195, February 1940.

⁴⁸ O'Brien, F. W., *New England Journal Med.*, 220: 917, 1939.

⁴⁹ Hedin, R. F., *Surgery*, 5: 161, 1939.

malignant. He stresses the importance of a good history, together with examination of the rectum and distal colon in suggesting the diagnosis. Sigmoidoscopic examination and double contrast stereo-roentgenographs establish the diagnosis. He states that drug therapy and dietary regimes are usually unsatisfactory and that x-ray therapy may cause improvement but does not cure, and recommends fulguration of all of the polyps within reach of the sigmoidoscope. For those beyond the reach of this instrument it may be necessary to fulgurate through a properly placed colostomy.

INTESTINAL OBSTRUCTION.—The Miller-Abbott tube has added a new chapter to the treatment of intestinal obstruction. Leigh, Nelson, and Swenson⁵⁰ report 76 cases of obstruction of the small intestine treated by this method. They use it in: (a) Paralytic ileus to deflate the bowel and to demonstrate the patency of the lumen, (b) in mechanical obstruction for deflation, diagnosis, and localization of the lesion, (c) for postoperative administration of food and fluids by mouth while the tube is serving the purpose of an ileostomy, and (d) for the control of postoperative distention in any type of case. They were able to intubate the small bowel in 68 of 76 cases. In these 68 cases in which the method was used they report a mortality rate of 5.9 percent as against 66.6 percent from 1916 to 1919 and 27.6 percent from 1932 to 1935. Reports from the Mayo Clinic⁵¹ are likewise good.

Wangensteen and Rea⁵² feel that the mechanical factor of distention and not a "toxic factor" accounts for the lethal issue in ileal obstruction. Norvikov⁵³ has found lumbar novocain block the most important conservative measure in the treatment of acute intestinal obstruction. He states that approximately one-third of all patients with this condition do not require operation and among them those with a dynamic ileus form the majority. A disturbance of innervation of the digestive tract is responsible for the dynamic type of ileus and therefore spinal anesthesia seems to be indicated from the pathogenic point of view. He, however, is opposed to spinal anesthesia in view of the possibility of grave complications and therefore prefers paravertebral block. If there are no signs of peritonitis, the patient is brought to the operating room and a right or left lumbar novocain block is performed. From 50 to 100 cc. of one-fourth percent solution is used. Of 139 patients with acute intestinal obstruction 63 were operated upon and 76 recovered after conservative treatment. In 46 patients the occlusion disappeared after employment of the novocain alone. He states that abdominal pains usually subside after 20 to 60 minutes, flatus is passed, and the abdomen becomes soft,

⁵⁰ Leigh, O. C., Nelson, J. A., and Swenson, P. C., *Ann. Surg.* 3: 186, February 1940.

⁵¹ Willson, D. M., *Proc. Staff. Meet. Mayo Clinic*, 15: 372, June 12, 1940.

⁵² Wangenstein, O. H. and Rea, C. E., *Surgery*, 5: 327, 1939.

⁵³ Norvikov, G. M., *Vestnik Khiv.* 58: 506, 1939.

palpation is painless, defecation takes place, and the patient falls off to sleep.

Fine and Gendel ⁵⁴ found that in experimental intestinal obstruction the intravenous injection of blood plasma in amounts adequate to replace that lost as a result of obstruction and distention of the empty small bowel, confers a protective influence sufficient to markedly prolong the life of the animal. Saline intravenously does not do this. Loss of plasma continues as long as distention continues in the obstructed small bowel.

APPENDICITIS ACUTE.—Many papers continue to appear on this subject and improvement in mortality rates has been reported from many quarters. Ochsner and Murray ⁵⁵ state that deaths from appendicitis are largely preventable. They describe acute appendicitis as inflammatory or obstructive, the latter being the more dangerous type on account of the frequent delay in making the diagnosis with resulting widespread contamination of the peritoneal cavity following rupture. An article by Kross ⁵⁶ on the matter of stump inversion deserves reviewing. He states that "although the clinical and pathological evidence favors the method of noninversion, the buried method is still used extensively." Experiments done by him on rabbits showed in the buried stump a combination of hemorrhagic infiltration and ulceration of the mucous membrane of the cecal wall and distal to the insertion of the purse string suture. In the unburied stump treated by simple ligation, there was an absence of these features. There also was frequent marked enlargement of the mesenteric lymph glands in which the stump was buried. Adhesions were present in all but were more marked in the ones where the stumps were buried. In several of the inverted stump experiments pin-headed cecal abscesses were found. C. W. Mayo ⁵⁷ stated in 1934, "Dr. Robertson in the Section of Pathologic Anatomy at the Clinic has found that invariably in the cases in which appendectomy with inversion of the stump has been done in combination with some other surgical procedure and death has resulted, there is a pus pocket in the inverted stump up to 21 days postoperatively." He also stated that he had had no occasion to regret not having inverted a stump.

Warren ⁵⁸ gives statistics on 111 perforated cases treated at the Peter Brent Brigham Hospital, Boston, from 1933 to 1937. Ninety-one patients were drained and 20 were not. Of those drained four developed fecal fistula while no fistula occurred in the nondrained. He summarizes his study by saying, "that a comparison of these two series shows in the undrained group a lower mortality rate and lower incidence of fecal fistula, but a higher postoperative reaction and

⁵⁴ Fine, J., and Gendel, S., *Ann. Surg.*, **112**: 240, August 1940.

⁵⁵ Ochsner, A., and Murray, S., *Amer. Jour. of Surg.* **46**: 566, December 1939.

⁵⁶ Kross, I., *Arch. of Surg.* **38**: 1016, 1939.

⁵⁷ Mayo, C. W., *Southwest. Med.* **18**: 397, 1934.

⁵⁸ Warren, R., *Ann. Surg.*, **110**: 222, 1939.

greater incidence of secondary abscess." He is of the opinion that in perforation without abscess, closure is safe and should be given a more extensive trial. Regarding drainage, Reid and Montanus⁵⁹ say, "in treating peritonitis of appendiceal origin we consider it wise to remove the appendix through a small McBurney incision and to drain or not, depending on the amount of necrosis and fecal contamination." Vale⁶⁰ has stressed the importance of frequent rectal examinations postoperatively and of transrectal drainage of abscesses in the rectovesical pouch.

Ravdin, Rhoads, and Lockwood⁶¹ reporting upon a series of 809 consecutive cases of acute appendicitis show a reduction in the mortality rate from 1.5 percent in the first 552 cases to 0.4 percent in the last 257. The improvement, they think, is due to the administration of sulfanilamide as there was no change in any other known factor. They use the drug in all drainage and borderline cases, and also preoperatively as an adjunct in the delayed method of treatment. They give it in 0.8 percent concentration in physiologic saline solution by hypodermoclysis. The usual dosage is 8 grams the first day and is reduced about 1 gram each day. Each day's dose is given in 4 installments at 6 hour intervals. If no unfavorable reactions occur, it is continued for 5 to 7 days. The blood sulfanilamide content should be maintained above 5 mg. percent and blood counts should be done every 1 to 2 days.

THROMBOPHLEBITIS.—In the opinion of Ochsner and DeBakey⁶² the old concept of blockage of the venous and lymphatic system in thrombophlebitis is wrong. Their clinical and experimental investigations lead them to believe that the symptoms are due to vasospasm of the arterial and venous systems and that the vasoconstricting impulses originate in the thrombophlebitic segment. Due to this vasospasm there is increased filtration pressure, relative anoxemia of the capillary endothelium and diminution in the flow of lymph, all of which increase the amount of perivascular fluid. By interrupting the vasoconstrictor impulses with procain hydrochloride infiltration of the sympathetic ganglions, there is produced a reestablishment of the normal exchange of intravascular and perivascular fluids. They treated 15 cases in this manner with prompt and permanent relief; 60 percent of these patients were discharged from the hospital as cured within 8 days after institution of treatment.

DeTakats and Jesser⁶³ say that 2 percent of all deaths, 6 percent of postoperative deaths, and 10 percent of all autopsies are due to pulmonary embolism. They are not in agreement with the concep-

⁵⁹ Reid, M. R. and Montanus, W. P., *A. M. A.* **114**: 1307, April 6, 1940.

⁶⁰ Vale, C. F., *Ann. Surg.*, **3**: 396, March 1940.

⁶¹ Ravdin, I. S., Rhoads, J. E. and Lockwood, J. S., *Ann. Surg.*, **3**: 53, January 1940.

⁶² Ochsner, A. and DeBakey, M., *J. A. M. A.*, **114**: 117, January 13, 1940.

⁶³ DeTakats, G., and Jesser, J. H., *J. A. M. A.*, **114**: 1415, April 13, 1940.

tion that the survival time following pulmonary embolism is too brief to establish therapeutic efforts, for in their group of 70 fatal embolisms, 8.5 percent of the patients died in less than 10 minutes, roughly 60 percent lived more than 1 hour, and 34 percent lived from 1 to several days. In a series of 100 patients, 87 died and 13 recovered. On the basis of experimental observations they believe that a widespread radiation of autonomic reflexes occurs during pulmonary embolism which may contribute to the cause of death. The vagus constricts the smooth muscles of the coronaries, the bronchi, and upper gastrointestinal tract. They suggest that atropine be used to block the vagus impulses and papaverine to relax the contracted smooth muscle. They give four predisposing factors to postoperative thrombosis: (a) Increase in the number of platelets which occurs following any major operation and reaches its peak between the 8th and 11th days, (b) increase in the fibrinogen, (c) a shift of the albumen-globulin ratio in favor of the globulins and, (d) an increase in viscosity. The increase in platelets and leucocytes results in liberation of thrombokinas and hastens the coagulation of stagnating blood adjoining an obstructing platelet thrombus. They stress the importance of clean sharp dissection and avoidance of trauma to the tissue. They also say that too little attention has been paid to counteracting the retardation of blood flow in patients after major operations and childbirth. Circulation may be improved by frequent turning in bed, deep breathing exercises, leg exercises by mounted bicycle pedals, digitalization of the failing heart, and by keeping away from the common semi-sitting position in bed which favors stagnation in the pelvic veins. When embolism occurs and cyanosis and dyspnea are the predominating symptoms, the patient should have 100 percent oxygen administered by the B. L. B. mask. In the syncopal type, characterized by pallor, fall in blood pressure and retrosternal pain without cyanosis, papaverine and atropine are indicated. Both of these drugs are given intravenously and should be dissolved in saline just previous to administration, papaverine in doses of $\frac{1}{2}$ grain and atropine in doses of $\frac{1}{60}$ to $\frac{1}{15}$ grain. They state that the successful removal of the obstructing plug in the pulmonary artery has been possible only 9 times in 132 attempts.

Very gratifying results are being reported upon the use of heparin in postoperative pulmonary embolism and in blood vessel surgery. By the intravenous administration of this substance the clotting time can be elevated to an arbitrary optimal level. Murray ⁴⁴ reports 440 patients in the hospital treated with heparin none of which developed thrombosis or embolism. Patients with thrombophlebitis were thought to be improved by the treatment and striking improvement was observed in a group of patients with pulmonary embolism.

⁴⁴ Murray, Gordon, Arch. Surg., 49: 307, February 1940.

Embolectomies were successful in 12 cases where heparin was used. McClure and Lam ⁶⁵ reported 8 patients with postoperative pulmonary embolism treated by general heparinization. All recovered, but one required a second course of heparin when a second infarction occurred 5 days after the first course was ended.

SURGERY OF LOW BACK PAIN.—The large number of papers appearing on low back pain testify as to the frequency with which this condition demands attention and to the stubbornness that it frequently shows in resisting treatment. After one reads these various articles he still is apt to be at sea when it comes to picking out the type of treatment that will give the patient the best chance for a permanent cure in the shortest period of time. Steindler's ⁶⁶ paper on interpretation of sciatic radiation and the syndromes of low back pain has helped a great deal in clarifying the situation. He has worked out 6 syndromes and gives a chart showing the trigger points for pain on palpation in these syndromes. By injection of these trigger points with novocain and getting relief from pain and signs he establishes the reflex character of the lesion which has much to do with suggesting the prognosis and treatment of the case.

Chamberlain and Young ⁶⁷ have found air myelography satisfactory in visualizing herniated intervertebral disks and other space-taking lesions in the spinal canal. They have used it in over 300 cases and in every case that came to operation the level of the lesion as predicted by the myelographic examination was verified at laminectomy. Camp ⁶⁸ reports a series of 203 cases in which laminectomy was performed and in which a roentgenologic diagnosis of protruded disk was made, the diagnosis was confirmed by the surgeon in 194 instances. His experience with the use of air as a substitute for iodized oil in cases of lesions associated with low back pain is that iodized oil is more accurate and that it will reveal certain lesions that air will fail to disclose. He states that further familiarity with the history and neurologic examination in protruded intervertebral disks indicates in a fair proportion of the cases the diagnosis and localization of the protrusion can be made clinically without resorting to any contrast agent.

Dockerty and Love ⁶⁹ pointed out the frequency of hypertrophy of the ligamentum flavum and stated that it is usually associated with a protruded disk so that the involved nerve root is not only compressed from in front by the protruding portion of the disk but also from the back by the pathologically enlarged ligament. Love ⁷⁰ says that 1.8

⁶⁵ McClure, R. D. and Lam, C. R., J. A. M. A., 114: 2065, May 25, 1940.

⁶⁶ Steindler, A., Jour. Bone and Joint Surg., 22: 28, January 1940.

⁶⁷ Chamberlain, W. E. and Young, B. R., J. A. M. A., 113: 2022, December 2, 1939.

⁶⁸ Camp, J. D., J. A. M. A., 113: 2024, December 2, 1939.

⁶⁹ Dockerty, M. B. and Love, J. G., Proc. Staff. Meet. Mayo Clinic, 15: 161, March, 1940.

⁷⁰ Love, J. G., J. A. M. A., 113: 2029, December 2, 1939.

percent of the patients seen by the orthopedic consultants at the Mayo Clinic because of low back pain and sciatic pain underwent laminectomy because of a diagnosis of protruded intervertebral disk. After having studied more than 300 cases of proved disk protrusions they have been able to formulate a symptom complex for the lumbar lesions. Given a patient with intractable low back and sciatic pain, spasm of the lumbar muscles, loss of normal lumbar lordosis, positive Laseque's and Kernig's signs, sciatic tenderness, and diminution or absence of the homolateral achilles reflex and the diagnosis is highly probable. If in addition there is a moderate elevation of the total protein content of the cerebrospinal fluid obtained on puncture in the lower part of the lumbar segment and there is narrowing of the 4th or 5th lumbar intervertebral spaces the picture is complete and the use of contrast media is not necessary. With the use of radiopaque oil, however, multiple lesions, which occur in about 12 percent of cases, will not be overlooked and shorter laminectomies can be used. Love prefers hemilaminectomy and in some cases is able to remove the protruded disk without doing a laminectomy. Hamby⁷¹ has found that in most of the cases with protrusion of disks of the 4th and 5th spaces, exposure is adequate without laminectomy, thereby saving time and conserving bone. In case hemilaminectomy fails to reveal a protruded disk most authorities think that a spinal fusion should be done. Many writers feel that lipiodol should not be put in the dural canal unless it is fairly certain that it will be removed by surgery at some later date. Bradford and Spurling,⁷² however, think that the use of 2 cc. is a safe procedure and that this amount is sufficient for adequate diagnosis. They insist that the diagnosis must be made clinically as well as roentgenologically if there is to be a successful selection of cases for operation.

HERNIA.—It has been only a few years since advocates of the injection treatment of hernia threatened to relegate hernia surgery to the land of forgotten operations. The statistics now being collected on this form of treatment show such poor end-results that it cannot be considered as a serious competitor of hernioplasty. Dobson⁷³ reports follow-up studies on 101 hernias in 74 patients followed for 6 months to 2½ years after the removal of the truss. There was a recurrence rate of 37.73 percent in the indirect and 68.42 percent in the direct inguinal hernias and 100 percent in the postoperative recurrent hernias. He is of the opinion that the injection treatment should be used only in small indirect hernias in patients with otherwise good abdominal structures who will not or cannot be operated upon. Penn Riddle,⁷⁴ who has recently published a book on the

⁷¹ Hamby, W. B., S. G. & O., 71: 344, September 1940.

⁷² Bradford, E. K. and Spurling, R. G., S. G. & O., 68: 446, October 1940.

⁷³ Dobson, L., Surgery, 7: 836, June 1940.

⁷⁴ Riddle, Penn, W. B. Saunders Co., Philadelphia, Pa.

injection treatment, reports 40 percent failures with this method of treatment.

There still is considerable discussion about suture material in hernia surgery and the silk technic seems to be gaining a good many converts; however, if the number of papers appearing in the literature is any criterion one must believe that fascial sutures are increasing in popularity. For inguinal hernias McCloskey and Lehman ⁷⁵ favor the McArthur type of operation which makes use of pedunculated strips of fascia from the aponeurosis of the external oblique muscle. Joyce ⁷⁶ reports 544 operations performed by the use of fascial sutures with a recurrence rate of 2.94 percent. Harrison ⁷⁷ repairs inguinal hernias by applying a patch of preserved ox fascia to the weak area from within the peritoneal cavity and claims that he gets permanent cures in both direct and indirect types.

Smith and Masson ⁷⁸ state that ventral hernias which they have not been able to repair previously can be repaired with sutures made from fascia lata. I would go further and say that this is also true of some inguinal hernias, especially those recurrent after several attempts to repair by other means. It has been my opinion for years that many of the poor results in hernia surgery are due to the fact that operations are carelessly done or that there has been improper selection of the types of operation. If the surgeon attempts to make one type of operation fit all types of inguinal hernias his recurrence rate is bound to be high. Fascial sutures do not handle easily and operations making use of them require additional time, which, in my experience, pays good dividends.

ORAL CYSTS OF DENTAL ORIGIN *

By Commander A. H. Yando, Dental Corps, United States Navy, and Lieutenant Carl A. Schlack, Dental Corps, United States Navy

During a regular extensive practice in oral surgery, many ramifications of dental cystic conditions present themselves. In this presentation of some of the cases associated with such conditions, it might be appropriate to review the general opinions concerning their etiology. Since the active parts of cysts are principally epithelial tissue, we have to deal primarily with the epithelial tissue, its derivation, and the circumstances under which it will proliferate.

Generally, cysts of dental origin may be classified as follows:

(1) Dentoperiosteal cysts, also called dental root cysts and radicular cysts, are those found apically or marginally, depending on the focus of stimulus (figs. 1, 2, and 3).

⁷⁵ McCloskey, J. F., and Lehman, J. A., *Ann. Surg.* 3: 611, April 1940.

⁷⁶ Joyce, T. M., *J. A. M. A.*, 115: 971, September 21, 1940.

⁷⁷ Harrison, P. W., *Surgery*, 7: 217, February 1940.

⁷⁸ Smith, C. H., and Masson, J. C., *Surgery*, 7: 204, 1940.

* From the U. S. Naval Dental School, Washington, D. C.

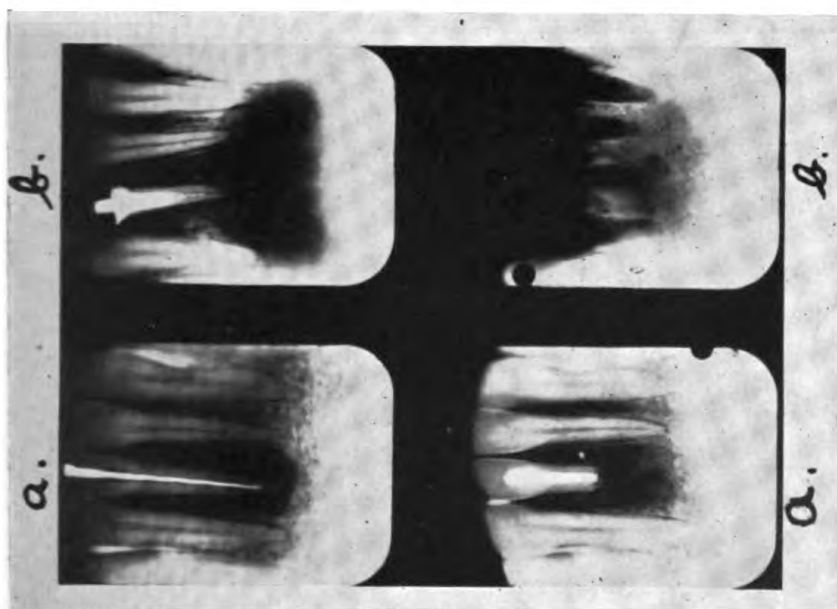


FIGURE 1.—A. DENTO-PERIOSTEAL CYST AND RESULTS OF APICTECTOMY AFTER 1 YEAR; B. DENTO-PERIOSTEAL CYST AND TREATMENT.

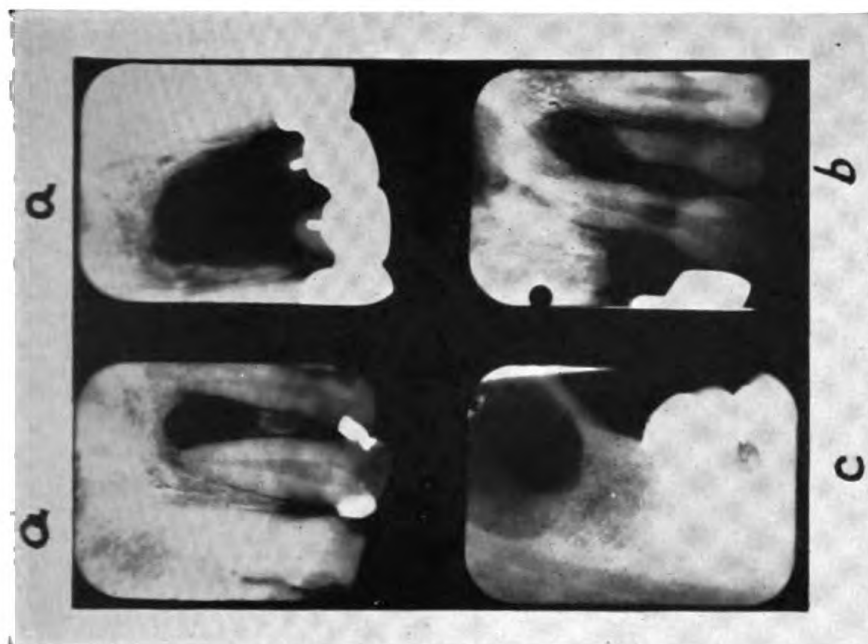


FIGURE 2.—A. DENTO-PERIOSTEAL CYST BELOW APEX AND TREATMENT; B. DENTO-PERIOSTEAL CYST. NOTE COMPLETE CALCIFICATION OF PULP; C. RESIDUAL DENTO-PERIOSTEAL CYST GROWN TO LARGE PROPORTIONS.

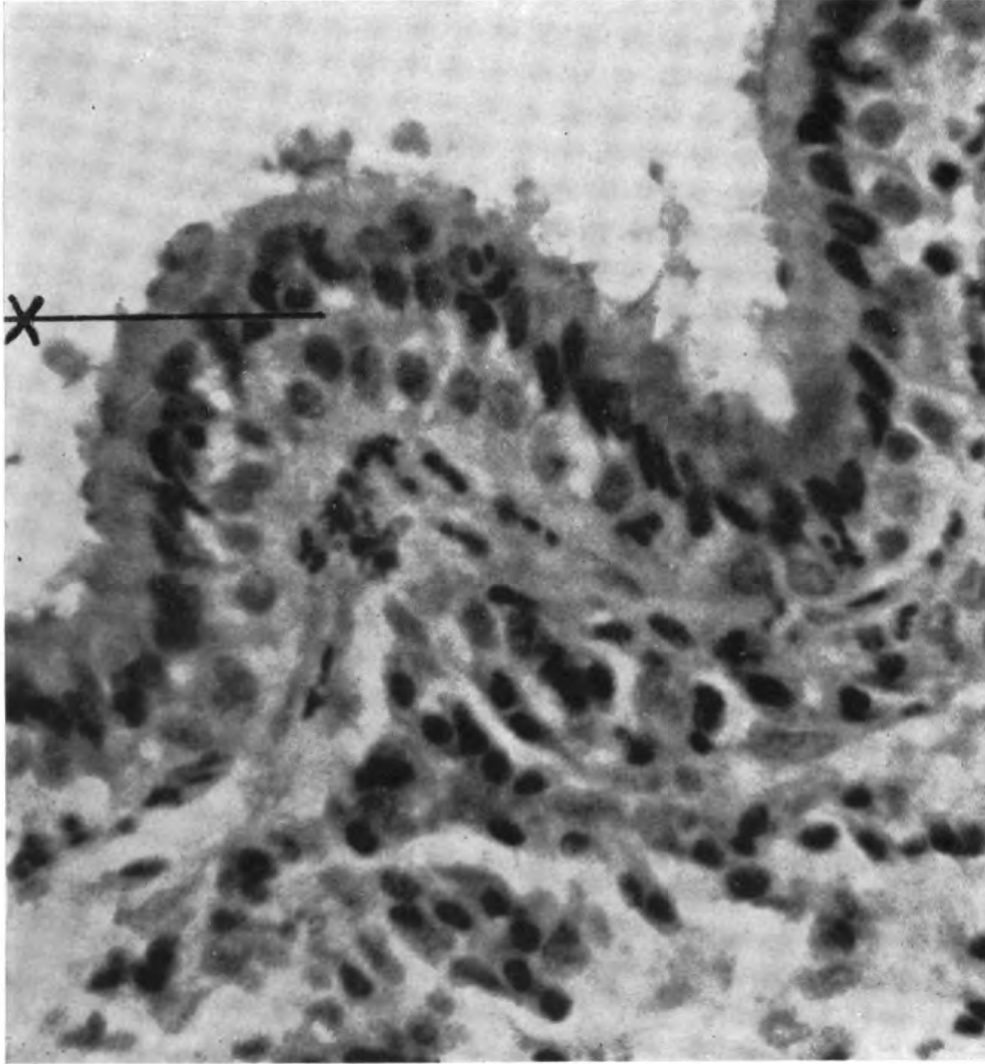


FIGURE 3.—WALL OF DENTO-PERIOSTEAL CYST. AT X WE SEE EPITHELIAL LINING COVERING UNDERLYING GRANULATION TISSUE.



FIGURE 4.—DENTIGEROUS CYST ABOUT IMPACTED THIRD MANDIBULAR MOLAR.

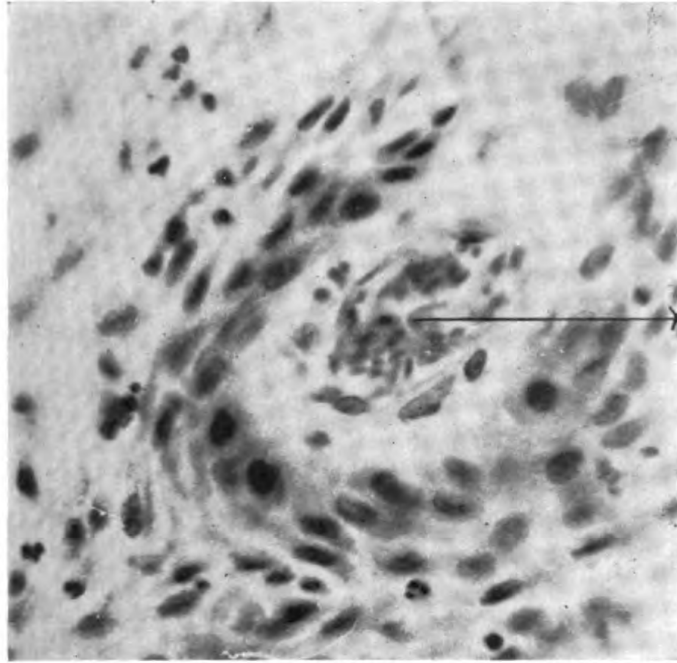


FIGURE 5.—A FOLLICLE OF A DENTIGEROUS CYST. NOTE BLOOD VESSEL AT X SURROUNDED BY EPITHELIUM OF CYST. COMPARE WITH FIGURE 6.

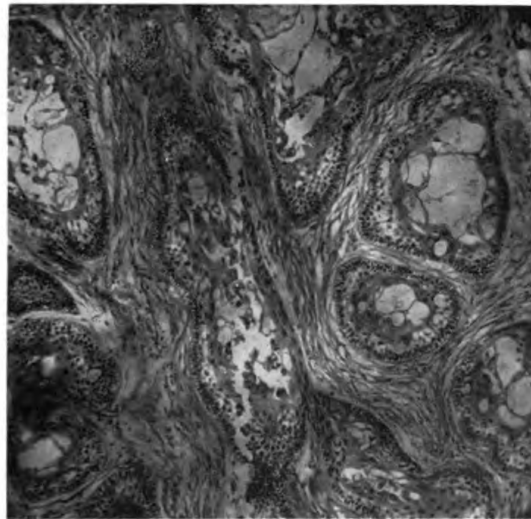


FIGURE 6.—PHOTOMICROGRAPH OF A CYSTIC AMELOBLASTOMA. NOTE ABSENCE OF BLOOD VESSELS WITHIN EPITHELIAL FOLLICLES AND THE SIMILARITY TO APPEARANCE OF ENAMEL ORGAN.



FIGURE 7.—A, MEDIAN ANTERIOR MAXILLARY CYST. TEETH WERE VITAL
B, RESULTS AFTER 6 MONTHS.

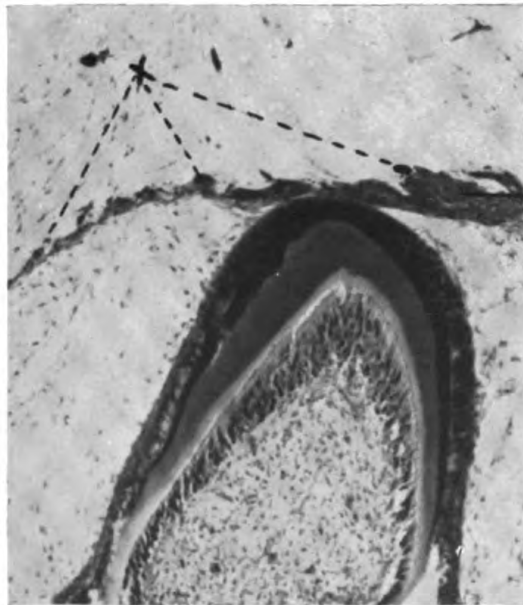


FIGURE 8.—DEVELOPING ENAMEL ORGAN OF MAXILLARY MOLAR OF HUMAN
FOETUS, $4\frac{1}{2}$ MONTHS. AT X WE SEE EPITHELIAL EXCRESCENCES OF THE
EXTERNAL EPITHELIAL LAYER OF CELLS OF THE ENAMEL ORGAN.

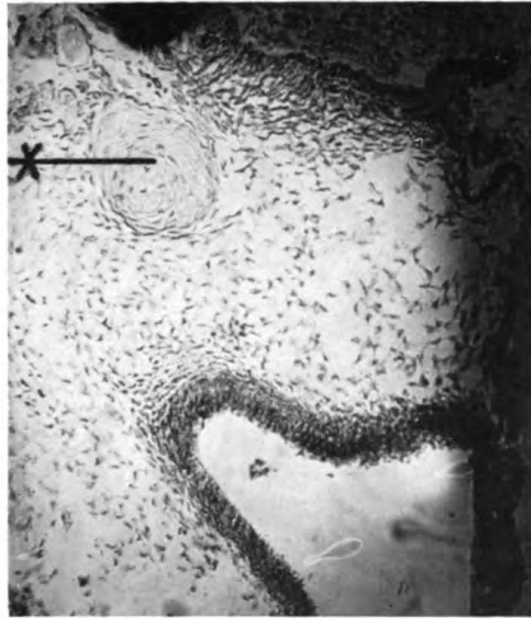


FIGURE 9.—ENAMEL ORGAN OF DEVELOPING MANDIBULAR DECIDUOUS CANINE OF HUMAN FOETUS, B. C. MEASUREMENT 11.5 CM. AT X AN EPITHELIAL COIL WITHIN THE STELLATE RETICULUM MAY BE SEEN.

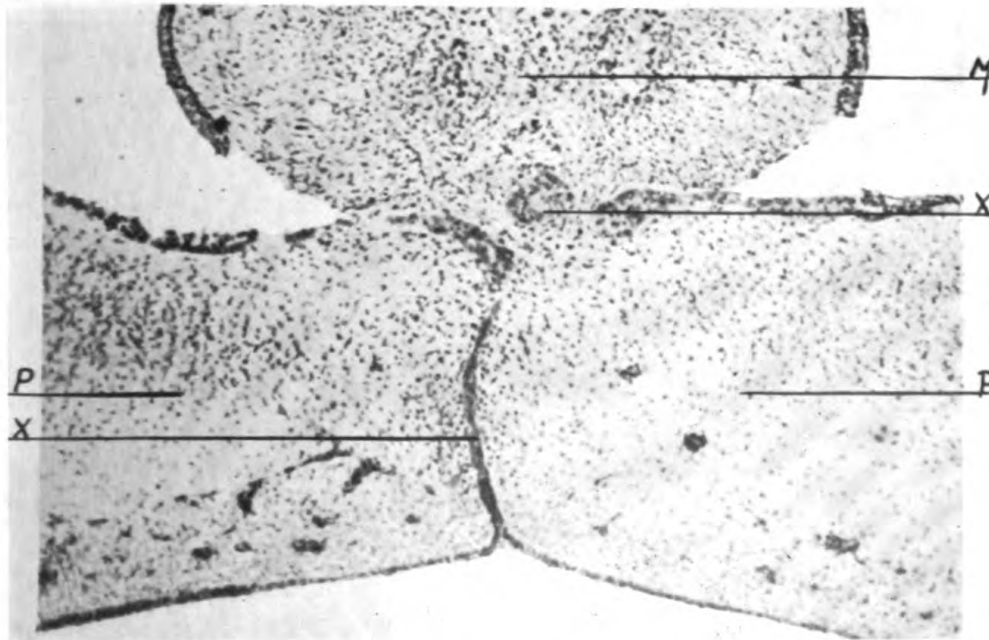


FIGURE 10.—SECTION THROUGH HEAD OF HUMAN FOETUS, B. C. MEASUREMENT 4 CM. MESIAL NASAL PROCESS M JOINING MAXILLARY PROCESSES P AT X. NOTE EPITHELIUM PRESENT WITHIN SUTURE. REMNANTS MAY GIVE RISE TO MEDIAN ANTERIOR MAXILLARY CYSTS.

(2) Dentigerous cysts or tooth bearing cysts, also called follicular or monolocular cysts, contain unerupted hard structures of teeth, normal or anomalous in development. This class also includes the cystic so-called odontomata and composite odontomata (figs. 4 and 5).

(3) Ameloblastomata or adamantinomata or multilocular cysts, which occur prior to the formation of the hard structures of the teeth (fig. 6).

(4) Cysts of pseudodental origin, the median anterior maxillary cysts, also called incisive canal cysts. These occur in the vicinity of the roots of teeth and appear to be of dental origin because of their position but may be of other origin and are frequently confused with dental cysts (fig. 7).

The epithelial tissues derived from the dentogingival lamina or tooth band, the external epithelial layer of cells of the enamel organ,^{1 2 3} and the internal or ameloblastic layer of cells of the enamel organ (fig. 8),^{3 4} may be responsible for cyst formation. The stellate reticulum and probably also the stratum intermedium of the enamel organ may likewise play an important role in the onset of such cystic conditions (fig. 9).

The dentigerous cysts are usually associated with the epithelial tissue derived from the external epithelial layer of cells of the enamel organ,^{1 2 3} from the dentogingival lamina, and probably also from the stellate reticulum.

The ameloblastomata are usually associated with the epithelial tissue derived from the internal or ameloblastic layer of cells of the enamel organ,² and possibly the stratum intermedium. Another source of epithelial structure from which the epithelium of cysts may be derived is that found in the dental periostium of the teeth representing remnants of the epithelial sheath of Hertwig, whose main function during development is that of guiding the development of the roots. Cysts derived from this epithelium exist in the greater percentage of cases and are the dentoperiosteal cysts usually associated with nonvital teeth.

The final source of cysts with which we deal are those of pseudodental origin or the median anterior maxillary cysts.⁵ The epithelial tissue may be derived from the epithelial remnants within the sutures formed by the union of the mesial and lateral nasal processes with the maxillary processes during development (fig. 10),^{5 6} and in some

¹ Lartschneider, J., *D. Cosmos*, 72: 239, 1930.

² Churchill, H. R.; *D. Cosmos*, 76: 1173, 1934.

³ Kantorowicz, A., *Klinische Zahnheilkunde* (Berlin, Musser, 1924), p. 252.

⁴ Robinson, H. B. J.; *J. Dent. Res.* 15: 334, 1936.

⁵ Kronfeld, R., *Histopathology of the Teeth* (Lea & Feb., Phila.) 1933, p. 406.

⁶ Grohs, R. J., *Journ. Dent. Res.* 14: 187, 1934.

cases representing rudimentary nasopalatine duct structures.⁷ Cysts derived from such tissue may be mistaken because of their position about the roots of teeth, as cysts of dental origin and oftentimes teeth may be needlessly sacrificed because of an evaluation of roentgenograms only (fig. 7).

Under suitable stimuli, activity of the cell rests or developing epithelial tissue is increased so that proliferation of the epithelial cells progresses to a point where insufficient nutrition may be received by the innermost cells and subsequent pyknosis, karyorrhexis, karyolysis and finally complete lysis of the cell takes place to produce the fluid contents of most cysts. These contents increase at a rate dependent upon many factors peculiar to the individual.

Stimuli in the case of dentoperiosteal cyst formation apically or marginally have been generally shown to be of infectious or traumatic origin.⁸ With the resistance of the individual and the attack of bacteria of a character resulting in granulation tissue formation, it has been shown how epithelial rests in the dental periosteum may proliferate not only in the immediate vicinity of the granulation tissue but likewise in rests some distance removed from the granulation tissue. This results in strands of epithelial cells growing towards the granulation tissue. Having reached the granulation tissue the epithelium proliferates locally and degenerates in a manner already described. Metabolism in the area of granulation tissue is increased as in all areas where such inflammatory processes are taking place. In these areas of inflammation all cellular activity is increased. We know that the fibroblasts proliferate too rapidly in acute inflammation to produce fibers which is their primary function. Only after the acute stage of inflammation has subsided do fibroblasts again assume the role of fiber formers. Any epithelial tissue which has been subject to a chronic injury of either chemical, traumatic, or infectious nature displays this proliferative potentiality, except that in some confined areas it degenerates and cysts may be produced as in the case of dentoperiosteal cysts. The question of malignancy from this type of cyst is considered almost negligible although a basal cell carcinoma in the wall of a maxillary cyst has been reported.⁹ Later, however, this carcinoma was found to have originated from the nasal epithelium rather than directly from the wall of the cyst.

Dental pulps need not always be nonvital for cysts to form at the apices of teeth as it depends, it seems, upon the reaction of the dental periosteum and the presence of epithelial rests that may or may not result in cyst formation as a reaction to injury of traumatic or infectious nature. The reader has already seen how the entire pulp may

⁷ Burket, L. W., *Arch. Path.* 6: 793, 1937.

⁸ Kronfeld, R., *Histopathology of the Teeth* (Lea & Feb., Phila., 1933) p. 157.

⁹ Pichler, H. J., *Journ. Dent. Res.* 14: 190, 1934.

be obliterated by the formation of either secondary dentin, pulp stone formation or both in response to some traumatic injury (fig. 2). Thus it can hardly be said that in such cases when cysts form they are due to a stimulus from an infectious process of the pulp which here must certainly have been very vitally reactive to the injury. It has been commonly seen how trauma may produce death of the pulp and how subsequent infection with a periapical response by the dental periosteum results in granulation tissue and probably cyst formation. Similar conditions prevail in almost any position of the parodontium if epithelial rests are present, granulation tissue is formed, and an avenue for bacterial invasion is present which, in the case of the marginal tissue, would necessarily be a broken epithelial attachment.

Formation of cysts about anomalous tooth formations as well as normally developing as yet unerupted teeth seems to originate from the cells of the external epithelial layer of cells of the enamel organ,^{1 3} as well as probably from cells of the stellate reticulum. Just what the stimulus in these cases is, however, is difficult to say. The external epithelial layer of cells of the enamel organ in tissue sections frequently shows epithelial proliferations here and there (fig. 8), and in the stellate reticulum we on rare occasions see epithelial pearl formation (fig. 9). It seems reasonable to attribute the origin of dentigerous cysts to these groups of epithelial cells. To these cysts has been given the potentiality of developing into ameloblastomata,^{10 11} although it has been shown how this conclusion may be a result of misinterpretation of tissue sections.² Finding of epithelial pearl formations in the stellate reticulum of ameloblastomata on rare occasions, has been described as similar to those found in squamous cell or epidermoid carcinoma.¹¹

Ameloblastomata or adamantinomata, also called multilocular cysts, seem histologically to be a result of ungoverned multiple enamel organ formations with proliferation of the ameloblastic layer playing a predominant role. Enamel is not formed but degenerative processes within the individual follicles may produce a soft fluid condition. The stimulus concerned with the formation of these cysts is vague as in the case of most tumors. It is this pathologic condition which has caused a great deal of concern to clinicians, surgeons, and pathologists. By 1936 about 357 cases had been reported in the literature.⁴ In 70 percent of the cases they were first noticed between 10 and 36 years of age. The earliest age was at birth.¹² The dentigerous cyst and the ameloblastoma have frequently been interpreted one for the other. Histologically, however, a differentiation has been shown;² compare figures 5 and 6. Both types of epithelial proliferation and cell characteristics have been found in the same mass of

¹ Cahn, L. R., *Dent. Cosmos*, 75: 889, 1933.

¹¹ Thoma, K. H., and Proctor, C. M., *Int. J. Orthod. and Oral Surg.*, 22: 308, 1937.

¹² Robinson, H. B. J., *Journ. Dent. Res.*, 15: 335, 1936.

tissue.¹³ Some investigators believe that they may arise from the walls of dentoperiosteal cysts as well as dentigerous cysts.^{11 14} Five percent of them have been reported to become malignant.¹⁵ They occur mostly but not always in the mandible at the angle¹⁵ and in the maxillae but have been found in the posterior wall of the pharynx, called hypophyseal duct tumors,¹⁶ and in the tibia.¹⁷ Concerning these last two locations the matter of interpretation as true ameloblastomata is still in a stage of conjecture. It is the practice as a safeguard in some clinics to irradiate the area from which an ameloblastoma has been removed because of the possibility of malignancy and recurrence.¹⁵ A differentiation has been made of ameloblastoma into an early, a later, and a mature type, corresponding to the various stages of development of the enamel organ.¹¹

The cysts here called pseudodental in origin are those which may arise in the vicinity of tooth roots but whose origin may be from those structures already mentioned. The stimulus responsible for their formation is obscure. In the case illustrated in figure 7, it was attributed to trauma due to rifle firing over a period of time.

Finally it is difficult indeed to determine clinically or roentgenologically whether we are dealing with a cystic condition in the vicinity of the teeth or not, whether we are dealing with dentoperiosteal cysts, dentigerous cysts, or ameloblastomata. This is especially true in the early stages of development of cysts. Consequently the study microscopically of such tissue removed from about the roots of teeth or other locations in the jaws cannot be emphasized sufficiently. It has been the practice here to send all tissues to the laboratory for sectioning and study in order to better acquaint ourselves with the condition that actually prevails necessitating surgical interference.

INJURY OF THE NERVOUS SYSTEM

COMMON PROBLEMS WITH RESPECT TO EFFECTS

By Lieutenant Commander George H. Hyslop, Medical Corps, United States Naval Reserve

INTRODUCTION

NATURE AND VARIETIES OF TRAUMA

Noxious external environmental stimuli which may affect the human body include abnormal temperature, electric shock, ultraviolet light, chemical substances, and mechanical or physical violence. The chemical agents may be gaseous, liquid, or solid.

¹³ Figl, F. A., and Stafne, E. C., *Journ. Dent. Res.*, 17: 328, 1938.

¹⁴ Carpenter, L. S., and Thoma, K. H., *Dent. Items Int.*, 55: 716, 1933.

¹⁵ Kotanyi, E., *Journ. Dent. Res.*, 15: 352, 1936.

¹⁶ Frazier, C. H., and Alpers, B. J., *Arch. Neur. & Psychit.*, 29: 935, 1932.

¹⁷ Baker, A. H., and Hawksley, L. M., *Brit. J. Surg.*, 18: 415, 1931.

Exposure to x-rays or radium emanation is a special variety of possible trauma, which may produce postirradiation fibrosis in tissues treated, and thus compress peripheral nerve trunks. The systemic effects of irradiation to certain areas of the body will consist of transient symptoms indicative of metabolic and vegetative nervous system upset. Occasionally one sees patients who present a picture of peripheral "neuritis," in the distribution of nerve trunks which have been recently exposed to large amounts of irradiation; this clinical picture likewise is transient and seldom is of a degree severe enough to produce definite objective signs of nerve trunk lesions. I have never seen a person in whom therapeutic irradiation produced any clinical evidence of a destructive lesion of the brain or spinal cord. These observations with respect to the effects of irradiation upon the central nervous system have been based upon 16 years' experience at the Memorial Hospital in New York City.

The following paper deals principally with the effects of mechanical or physical violence affecting the central nervous system.

SIGNS OF INVOLVEMENT OF THE NERVOUS SYSTEM

The effects of trauma upon the nervous system may be precisely localized or focal, diffuse, or disseminated. The nature of the trauma is the chief determinant of the distribution of lesions resulting from it.

For purposes of convenience, the nervous system may be divided into 3 portions—the intracranial portion and the cranial nerves, the spinal cord, and the peripheral nerves. The central vegetative nervous system alone, comprised of the autonomic and sympathetic divisions, is not directly affected by trauma except that disorders of the vegetative nervous system may accompany certain psychological phenomena which may be related to injury, or apparently result from damage to the cerebral vegetative centers in cases of brain injury.

There are certain commonly known and readily observed "localizing" signs of involvement of the nervous system which enable a rough diagnosis as to where lesions exist. Such symptoms or signs enable one to center attention upon the degree or character of abnormality in the particular portion of the nervous system affected.

1. *The brain and cranial nerves.*—Impairment of intelligence, speech defects (aphasia and also enunciation and pronunciation disturbance); weakness and impaired sensation of hemiplegic character (face, arm, and leg on one side); impairment of cranial nerve function, smell, vision, taste, hearing, and movement of eyeballs, face, throat, and tongue muscles.

2. *The spinal cord.*—Atrophy and weakness of limb and trunk muscles, the muscles involved depending upon the level of the spinal cord affected; the same holds true with respect to the areas of impair-

ment of sensation; impairment of bladder and rectal sphincter control, and sex function in the male.

3. *Spinal nerve roots and peripheral nerves.*—Atrophy, weakness, and impairment of sensation in tissues supplied by nerve roots or trunks involved. Usually, one limb or a portion of a limb is found affected. The most common findings are related to the upper arm and shoulder movement, hand and finger movement, and in the lower extremity the presence of foot drop and also the "sciatica" picture.

In considering the effects of trauma upon the nervous system, the problem divides itself into two types of situation—first, in which the nervous system or body as a whole is healthy and normal at the time the trauma occurs, and second, in which the nervous system or body as a whole is abnormal in some manner due to local or systemic disease. I use the word "disease" as implying some physiological, structural, or even psychological disorder, whether or not the individual is aware of its existence.

The system of standard nomenclature classifies disease in terms of causal agent or etiology, and the part of the body affected. Thus the classification is etiological and anatomical.

Diseases of the nervous system may be physical or mental. Physical diseases may arise out of developmental defect, infection, intoxication, injury, tumor or neoplasm, and two special classes of disorders in which the etiology is not thoroughly understood—the groups of degenerative and ill-defined diseases—but in which structural defect is manifest.

Mental diseases are classed as the psychoses, reactions of psychopathic personalities, the mental defects, and the psychoneuroses. The various causes of physical disease mentioned above apply also to mental disorders. Furthermore, mental disease may or may not be associated with or due to structural changes in the brain. The "organic" mental diseases are those in which there is actual disease in or defect of brain tissue. The other mental diseases are called "psychogenic." However, manic-depressive psychoses, constitutional psychopathic states, and schizophrenia or dementia praecox, while termed "psychogenic," are disorders which have a constitutional or hereditary basis which is strong enough to show itself in the personality of the individual before the onset of his psychosis, which takes a more or less characteristic form. The truly "psychogenic" mental disorders are characterized by the psychoneuroses, in which inability to adapt advantageously to experience leads to disturbances of feeling and thought.

HISTORY AND PHYSICAL EXAMINATION

In examining a person who has been injured, both an adequate history and a competent physical survey are necessary. The degree

of detail required will depend somewhat upon the circumstances or nature of the alleged disability. A person whose ulnar nerve has been severed and who is in apparent normal health in every other respect does not require a very detailed study. In an individual sustaining generalized brain injury who happens also to require an evaluation of previous systemic health and a psychiatric survey, the situation is complex and the physician has much to do. In an individual who has had insignificant physical trauma, but who presents a problem in mental disorder, a diagnosis may have to be reserved until many hours have been spent in gathering relevant information about the patient's personality and his life experiences. A patient may have to be studied in hospital before a diagnosis can be made.

These principles hold true in ordinary medical practice. In medico-legal work, an accurate and detailed history of the manner of injury, its immediate effects, the treatment given, and the course of the disability have a particular importance. An experienced physician will often know when to discount the history on the score that the patient is telling a story which is improbable and may be motivated by purposes which lead either to conscious or unconscious distortion of facts. The manner in which the patient relates the story of disability may be of importance in evaluating subjective symptoms and also the degree of handicap alleged.

It is a truism that a particular disease or disability has its own normal "life history." Therefore, an injured person whose history and findings on examination do not conform to a well-known physical disease entity due solely to trauma, should arouse the attention of the examiner to the possibility that important information has been withheld, or may be brought out by further inquiry.

In getting a history, it is always necessary to have a clear understanding as to what the patient means by the words he uses to describe his condition or symptoms. Patients are prone to use words or terms which have a diagnostic import, and which imply that the patient has come to certain conclusions about his experiences. There are certain terms which may mislead the examiner unless he is careful.

"Weakness" may be alleged. Whether the patient means fatigable, lacking in energy, disinterested in making any effort, short of breath on exertion, or avoiding activity for fear of certain specific untoward consequences must be determined. The word "weakness" may be used to describe spasticity in which there is no actual weakness; the word is frequently used by a person who is not weak in the sense that muscles are paralyzed, but is merely clumsy or awkward, or perhaps handicapped by impairment of equilibrium.

"Spells" or "fainting spells" may be alleged. If a patient is properly questioned, one can distinguish between episodic symptoms which may represent anything from attacks of transient vertigo,

through the syncope which occurs in certain types of circulation disorders, to genuine epilepsy or even hysterical tantrums.

"Dizziness" is a word frequently used to describe certain feelings which do not in any way implicate the vestibular apparatus, or the cerebral control of equilibrium. Sometimes a patient with locomotor ataxia who has trouble standing and walking because of damage to spinal cord nerve tracts will use the word "dizzy" to describe his defect or lack of balance and coordination in getting about. To the observer, this patient may move about in a way very similar to that of the individual with a cerebellar tumor who actually does experience vertigo. Transient impairment of focus of vision as well as certain types of subjective visual defect may be accompanied by vertigo and the patient may lump the concatenation of symptoms under the term "dizziness," with the result that the doctor does not give attention to the visual symptoms which may be important in making the diagnosis.

Another term which should be investigated is "headache." There are many kinds of pain in the head. Its location, frequency, duration, and whether or not its appearance is influenced by or correlated with certain circumstances or other symptoms, will be important in judging the probable character of the discomfort alleged. The same principle is true of pain or discomfort anywhere else in the body. If a discomfort, when described in detail, conforms to some known physical ailment, the diagnostic problem may be simplified. When the description very obviously implies physiological impossibilities or seems to be the product of conscious or unconscious elaboration, one is put on guard. One is familiar with the combination of "headache and dizziness" which often is accepted by an examiner as evidence of organic or physiological residuals of a cerebral concussion, and consequently the rating of an individual as to some degree disabled by a definite physical disorder.

The terms "concussion" and "unconscious" may be used loosely. While it is true that competent physicians do not use the term "concussion" with uniformity as to its implication, one generally thinks of a person suffering from concussion as having a collapse in which there will be a fall to the floor or ground and complete loss of consciousness even if only for a few seconds. The word "concussion" may or may not imply a particular type and degree of brain reaction to injury. While one is familiar with the fact that a blow to the head may result in collapse and loss of consciousness, and also cause microscopic lesions, particularly in the brain stem, it is not true that collapse and loss of consciousness inevitably mean that microscopic cerebral lesions have occurred. The reverse situation also should be kept in mind. There are instances of direct and indirect violence to the head in which there is no collapse and apparently no alteration of consciousness, yet the violence may result in not only microscopic but gross

damage to the skull and its contents. It is important also to keep in mind that alteration of consciousness may be as significant as complete loss of consciousness and collapse. Consider, for example, the individuals who sustain a blow to the head and continue their activities for from a few moments to hours and appear to be uninjured, yet are amnesic with complete loss of memory for the events and activities of this period as well as the actual injury itself.

I have indicated the importance of obtaining an accurate and detailed account of a person's injury and the things which follow it. Proper allowance should be made for the normal forgetfulness which will lead to inaccuracies of statement, especially when a history is obtained long after an accident. However, the character and quantity of inaccurate statements will often show what the patient's attitude is and whether or not the inaccuracies are deliberate and in the direction of deceiving the examiner. In compensation cases, the experienced referee may appreciate the facts as to inaccuracies of statement if they are brought to his attention. In court cases, it may be more difficult to make the situation clear to the average jury, whose members have little or no knowledge of medicine.

Information from physicians and hospitals previously in charge of the patient may be important. Original hospital records are infinitely more useful than abstracts or summaries.

In physical examination of the patient the physician should, as far as possible, have his procedure so devised that findings are not influenced by the cooperation factor. There are indirect means of testing for functions, without suggesting responses. Sometimes it is necessary to alter one's examination routine. In a neurological examination one may be accustomed to first test the motor system, then the reflexes, then sensation, and then the cranial nerves. Sometimes, following this particular order may be improperly suggestive to the patient, especially if he has been through a similar examination previously and has in mind what comes next and what his responses were on the previous occasion. One has to use one's judgment in deciding when and where to modify one's routine—the kind of individual one is dealing with is sometimes revealed during the taking of the history, and the presenting complaints are factors in judging what the procedure shall be.

One still sees skull films which are utterly inadequate. Not only may they be poorly exposed and developed, but the films made do not give any information about regions of the skull which may be important. Some x-ray men never take stereoscopic films. I believe that stereoscopic films both in the lateral and anteroposterior planes are a minimum routine requirement. Sometimes additional films are necessary to define pathology clearly. In x-ray examination of the extremities it may be desirable to take films of the uninjured

limbs. Calcifications in the region of an injured shoulder joint may not be responsible for any symptoms, and the presence of similar calcifications in the uninjured shoulder which is free of symptoms will reduce the risk of error in clinical interpretation. In x-ray study of the spine, certain types of injuries may necessitate taking stereoscopic films. The roentgenologist ought to confer with the examining physician in deciding what views should be taken. Otherwise, the roentgenologist works by rule of thumb and may fail to take the proper films.

DIAGNOSIS

After one's examination has been completed, one is concerned with the problem of a correct diagnosis. I know of no way which is going to guarantee correctness of diagnosis when one has to deal with a large number of complicated facts whose relations may be difficult to evaluate. In such cases the physician's particular bias and scientific interest may lead him to select certain portions of the evidence as important and crucial. Another equally competent physician may come to a dissimilar diagnostic conclusion. However, discrepancies in opinion will be at a minimum when diagnosis is based upon complete information and consideration of all the facts.

CAUSAL RELATIONSHIP

It is after one has made a diagnosis that the problem of causal relation has to be considered.

The various kinds of cause and the meaning of the concept "cause" should be based upon principles of logic and science. State labor departments in some instances have set up standards of "causal relationship" which may not be in accordance with medical science. Referees and other administration officers may be guided by either a sociological theory or certain theories as to procedure, rather than medical advice—including even at times the opinions of State department medical examiners.

In the adjudication of claims for disability arising out of the course of employment, confusion often arises because of the failure to distinguish properly between true cause and either a contributing or an accidental cause. There may be a complex array of facts, of which only one is the true cause and of basic importance. There is difficulty especially in situations where an injury or occupational exposure is alleged to have either precipitated or aggravated the course of some independent and pre-existing condition which has no true causal relationship whatever. Sometimes the disability first manifests itself within a few days after some injury.

One of the most satisfactory methods of determining causal relationship in certain types of disability is access to reliable statistics.

Properly gathered, statistics will decrease the number of opportunities for alleging that because we do not positively know the cause of a particular disease, we are unable to exclude certain possibilities as to causal connection.

Prompt and thorough recording of facts may have crucial importance in complex cases, in which the manner or degree to which injury is actually responsible for disability is to be determined.

TRAUMA TO THE HEAD

Violence may be direct or indirect. Indirect violence is most frequently associated with a fall during which the individual lands on the feet or buttocks in such a way that the physical force is transmitted through the spinal column to the base of the skull. Fracture of the base of the skull, and damage to meninges, brain, and blood vessels may result.

Direct trauma is the most frequent cause of injury to the head.

The results of trauma to the head may be classed on a temporal basis, as immediate or delayed.

IMMEDIATE RESULTS OF TRAUMA TO THE HEAD

1. *Fracture of the skull.*—This is always immediate if it occurs at all. The type of fracture depends partly upon the condition of the skull, the age of the patient, the thickness of the skull, and whether the bone of the skull is diseased in any way. The other conditioning or determining factor is the amount of violence, the direction from which it comes, the portion of the skull struck, the size and shape of the striking object. The fracture may be simple or linear, fragmented or comminuted, depressed, or compound. Certain types of fractures are apt to occur at the point of the skull opposite to the area actually struck. In most instances, x-ray examination will reveal fractures of the vault. Fractures of the base sometimes cannot be revealed by x-ray examination but are with practical certainty judged to be present on the basis of certain types of associated injury to soft parts and such symptoms as certain types of bleeding from the cranial orifices and the persistent escape of spinal fluid either through the nose or the ear. It should be kept in mind that an eardrum may be ruptured and blood escape from the external auditory canal without any fracture to the bones about the ear, and that injury to the nose may occur without fracture of the base of the skull and cause prolonged bleeding from the nose and epipharynx. Likewise, bleeding from the mouth may be due to injury to the teeth and gums.

One must always keep in mind the type of injury and whether it is a possible or probable cause of a fracture found.

2. *Concussion*.—This term is used very loosely. Essentially, it implies the immediate reaction of the brain to violence and traditionally this reaction is characterized by falling or collapse and some alteration of consciousness. The duration of a total loss of consciousness and collapse will vary roughly with the actual damage to the brain and in cases where brain injury has been severe, one would find the signs of "shock." Whether there is always some structural lesion to the brain is still a matter of speculative argument. The great majority of individuals who sustain a concussion never come to autopsy; therefore, argument is primarily by comparison or analogy. Many physicians assume that even a mild concussion is accompanied by some rupture of tissues, small capillaries and their walls, interstitial elements of the brain, and even brain cells. They also assume that there is a disturbed innervation of the cerebral blood vessels.

Individuals who have sustained a concussion must be examined with thoroughness at the earliest opportunity. A careful neurological examination may reveal definite although slight objective evidence that the head injury has produced a focal reaction which does not produce any symptoms of which the patient is aware. Pupil changes, abnormalities of extra-ocular muscle movement, and alterations of the superficial reflexes should be searched for carefully and repeatedly during the first 72 hours after a concussion. The discovery of such objective signs of focal brain reaction and their duration, if found, are infinitely more important than the mere fact of a loss of consciousness. If such signs are definite and persist for several days, they may very well indicate that some of these assumed microscopic structural changes actually occurred in the particular patient. However, my experience is that although such transient signs are fairly common, they do not necessarily imply any persistent disability or sequelae of "postconcussion" character.

3. *Injury to the brain additional to concussion*.—Any tissue in the body may be "bruised" by physical violence. Nature tends to bring about recovery. Uncomplicated contusion or "bruising" of the brain with attendant swelling but no evidence of hemorrhage apparently is subject to the same beneficent effects of nature and complete clinical recovery is frequent. The severe inflammatory reactions in the brain which occur in the course of infections of the nervous system, as well as in certain toxic reactions and which result in full recovery, indicate caution in assuming that similar tissue change due to injury must produce permanent damage and disability.

The brain substance may have multiple lacerations and in unusual instance there may be gross intracerebral bleeding. While contusions and small lacerations may be directly beneath the site of the blow, or at times contrecoup, severe head injuries may be responsible for such lesions almost anywhere in the brain. The frontal lobes are

particularly vulnerable, partly because there is free space about them, but especially because the base of the skull in the frontal region has irregularities and sharp edges against which the brain may be "jounced." When there has been widespread damage to the frontal lobes, some degree of mental impairment or deterioration is common. Such mental changes will in my experience be manifest within less than 6 months after an injury and frequently the injury has been followed by prolonged unconsciousness and then a period of acutely altered personality which may persist for days or several weeks before it apparently subsides. This immediate mental sequel of head injury is known as a posttraumatic delirium or posttraumatic acute psychosis.

4. *The meninges.*—Rupture of the meninges may take place at points in contact with fracture of the skull. The dura is usually involved in such instances. The dura may be pinched in a fracture, and delay union as well as cause certain localized pain. Without rupture of the dura, there may be demonstrable lesions in the arachnoid and pia, resulting in adhesions between the meninges and the cerebral cortex and even loculations of spinal fluid which may cause local effects comparable to those resulting from any expanding lesion.

5. *Blood vessels.*—Intracranial blood vessels are either intra- or extracerebral. The intracerebral blood vessels, when damaged, may cause some degree of extravasation of blood into the surrounding tissues. In severe concussions or greater injuries, it is believed that often there is transient paralysis of innervation of small arteries in the brain substance, particularly in the mid brain and medulla, without necessarily any rupture of the walls of these vessels. There may be some diapedesis. In such cases, it is thought, there may be permanent impairment of innervation of the affected blood vessels and thus one accounts for some instances of the postconcussion syndrome, particularly the positional vertigo. Capillaries naturally are broken where there are lacerations of the brain substance. Gross intracerebral hemorrhage occurs only when there have been large lacerations of the brain substance.

The extracerebral blood vessels include those of the meninges and the major vessels entering or leaving the skull at the base. Injury to the larger arteries entering the cranium at the base will result in immediate death. Rupture of the large veins leaving the cranium at the base will practically always result in death, whether or not there has been a severe head injury with widespread and severe damage to the brain. The exception here is in instances of laceration of vein walls by fracture fragments with prompt operative intervention.

The vessels of the meninges present an interesting and not by any means rare problem. Rupture of a branch of the middle meningeal artery causing epidural hematoma is usually accompanied by a tear

of the dura, whether or not there is an overlying skull fracture. In practically every instance, the typical clinical picture is acute with a rapid development of coma and paralysis of hemiplegic type.

Subarachnoid bleeding is not infrequently an accompaniment of fracture of the base of the skull and is also common with injuries causing severe contusion or small lacerations of the brain substance.

Subdural bleeding is most frequently from veins. The patient whose blood vessels are fragile from arteriosclerosis or other constitutional cause is predisposed to the development of subdural bleeding. It is well known that even apparently very slight head injury may result in subdural bleeding. In a large percentage of cases of subdural hematoma, there is a delay of weeks or longer before the definite development of symptoms, signs and disability due to what by then is a "chronic subdural hematoma." Surgery is the only treatment for a subdural hematoma.

LATER OR DELAYED RESULTS OF INJURY TO THE HEAD

Sequelae of head injuries can be classed as on a physical basis and due to structural changes, to possible disorders of cerebral vasomotor balance without demonstrable structural changes in brain tissue, or to purely psychogenic disorders. The term "posttraumatic encephalopathy" should, I believe, be reserved for cases in which symptoms are based on physical effects of injury to the brain.

1. Personality changes which are the product of structural disease may at times be quite clearly organic in character. The changes in behavior and attitude which are not on a structural basis may be due to the patient's reaction to certain subjective symptoms which may be on a physical basis. The purely psychoneurotic disorders occur in individuals to whom their accident or injury has some special meaning. The reactions vary from the conversion hysterical at one end of the scale down through anxiety states to the litigation or compensation neurotic response. The degree of conscious distortion of facts is in proportion to the financial significance of the injury in persons who are in the litigation-neurotic group; sometimes the litigation motive is not present to any determinable degree, but the symptoms alleged may be advantageous in connection with domestic or other personal matters and consciously produced if not distorted for such ulterior purposes. The true psychoneurotic believes in his symptoms, and his behavior will in general be found consistent with the symptoms alleged, although it is not uncommon to observe some contradictions under circumstances which show that attention has been temporarily distracted from the symptoms. The individual whose behavior is grossly and persistently in contradiction to symptoms alleged, and who will display a degree of disability during examination or under circumstances which make it evident that an attempt is being made

to impress those present, and then resumes normal behavior as soon as interested witnesses are not present, is consciously simulating. The fraud may often falsify seriously in the direction of exaggerating the severity of the injury when it occurred, and in describing symptoms which followed the injury. Malingering is seldom engaged in by the individual who has a normally developed personality. Not rarely, what commences as malingering may, if the patient is sufficiently psychopathic, be a prelude to actual mental disease.

Organic posttraumatic psychoses due solely to structural damage from injury are rare, and follow severe damage to the brain at the time of the injury. The clinical picture may be that of mental deterioration with alteration of character and defects of emotional control. Patients with such mental disorder will develop definite symptoms in most instances within 6 months after an injury and to regard the injury as solely responsible one should assume no accompanying independent organic disease of the brain. From my experience with such cases, I believe that there are definite, even if slight, mental changes from the time of the injury and that the more obvious symptoms arise as repair processes and scarring reach their maximum. These patients are incurable in the light of our present knowledge. Certain operations on the frontal lobes, which are still in the experimental stage, might be justified if the patient and responsible relatives have no objection to the operation, which might reduce the symptoms of emotional instability.

Frequently, the question arises as to whether a so-called constitutional psychosis such as dementia praecox is caused by a brain injury. Academically, if there has been a really severe brain injury with definite clinical evidence of traumatic lesions, an episode of constitutional psychosis might develop. Without such severe traumatic pathology, a brain injury which seriously disturbs a patient's manner of living and furnishes conflicts and problems of adjustment which are too much for a constitutionally defective individual to handle, may precipitate a psychosis. The mental mechanisms in such cases are analogous to those which bring about one or the other kind of psychoneurotic reaction, and the injury is seldom more than a minor contributory precipitating cause and does not alter the course of the psychosis.

I have seen a number of individuals in whom I was able to obtain sufficient history to make clear that there was actual mental disease prior to the injury and that the injury was neither severe enough to produce any real brain damage nor provide a situation which altered the natural development of what had preceded the injury. In such cases it can be maintained that the injury has had neither precipitating nor aggravating influence upon an independently existing mental disorder.

2. The so-called "postconcussion syndrome" may occur without any clinical or otherwise demonstrable damage to the brain. Pain in the head, impairment of equilibrium, transient indistinctness of vision, and discomfort on changes of body position, particularly in the direction of stooping forward, are common symptoms after a head injury which has produced a concussion reaction or damage either to the skull or focal damage to the brain. In the great majority of instances, such symptoms spontaneously disappear. In an otherwise healthy person, such symptoms as a rule clear up within 6 months. They may last from a few days to 2 or 3 weeks after a moderate concussion reaction and are protracted in general in proportion to its severity.

The head pain following brain injury, whether or not a focal lesion is present, may vary both as to location and duration. In general, the postconcussion headache will be influenced by fatigue, emotional tension, and activity involving stooping or lowering of the head. If severe and frequent, the pain is apt to interfere with sleep. Hot and humid weather frequently aggravates such headache.

Head pain may be due to bruising of the skull, to adhesions between the meninges and either the skull or the brain, or to focal lesions outside the cerebrum. Such pain is apt to be localized. Wilder Penfield and others, dealing with patients suffering from conditions not the result of injury, have demonstrated that head pain which is persistently referred to a particular area may be due to transient changes in the caliber of meningeal vessels.

If a patient who has had a head injury describes the head pain in a different way each time he is examined, or if he links the occurrence of pain with symptoms which are inconsistent with any known genuine physical condition, it is probable that the head pain in such an instance is entirely of psychogenic origin although there may at one time have been head pain which had a physical basis. Whether, in such a case, one is dealing with malingering or a psychoneurosis may depend upon cumulative or other evidence.

One must question carefully as to what a patient means when vertigo or dizziness are alleged. Central or peripheral vestibular pathway damage will produce an impairment of equilibrium in association with subjective or objective vertigo.

Fatigability and irritability, not due to psychoneurotic reactions, are common.

The exact mechanism of these particular symptoms is not definitely known. An irritability or instability of the vasomotor centers and perhaps damage to blood vessel innervation has been suggested as a physical basis for the symptoms.

As a consequence of such microscopic lesions as are assumed to occur with concussion, it is argued that nature's reparative process is not perfect and there are residual microscopically demonstrable scars

which provide a structural basis for the chain of symptoms termed the "postconcussion syndrome." Such lesions have been demonstrated in individuals who have sustained severe cerebral injuries leading to death. They have also been described as occurring in individuals subject to long-continued and repeated head injuries with concussion. The clinical entity seen in pugilists, known as "punch-drunk" is illustrative.

There are three points to be kept in mind in connection with the above assumptions and analogies. First is the fact that collapse and loss of consciousness identical with "concussion" may occur in the absence of any violence to the head. The knock-out from a solar-plexus blow, or from certain blows to the neck in ju-jitsu, an ordinary syncope in people with vasomotor instability and even fainting from fright, show that a disturbance in function of the medullary vasomotor centers may be sufficient to produce a symptom complex indistinguishable from slight "concussion." Therefore, it is quite possible that a certain proportion of concussions due to head injury are of vasomotor character with transient medullary reaction and do not imply actual destruction of cells or other tissue within the brain or any other permanent effect. Second, if one is going to depend upon argument by analogy, one should not maintain that the assumed microscopic lesions in certain cases of concussion are necessarily the basis for any particular symptoms. It is well known that injury may cause severe structural changes, without the occurrence of "postconcussion" symptoms. Other types of lesions occur, without such symptoms. Individuals subject to repeated attacks of grand mal epilepsy are known to have widespread microscopic and even grossly demonstrable brain lesions, yet it is rare to find an epileptic who complains of "postconcussion" symptoms between his attacks. Individuals with active syphilis of the brain, syphilitics with meningeal or vascular cerebral disease who have been treated, individuals who have generalized and cerebral arteriosclerosis, people who have had a severe apoplexy and even individuals with certain chronic degenerative diseases of the brain such as multiple sclerosis, do not as a rule complain of "postconcussion" symptoms. Third, the assumption that the postconcussion symptoms are due to cerebral circulatory instability should not be regarded as proof that such instability is the result of structural lesions or even permanent disturbance of innervation of the cerebral vessels due to injury. Almost identical symptoms are observed frequently in uninjured individuals whose basic disorder is entirely psychoneurotic but who manifest reflexly a circulatory instability which will persist as long as the psychoneurosis continues—even for years.

I feel that judgment as to the nature of postconcussion symptoms must be the result of attention to all the relevant facts in each patient under study. Whenever a judgment has to be rendered in the case of

a patient whose symptoms are not corroborated by a careful physical and neurological examination, one must search for evidence of psychiatric importance, in connection with a possible neurotic or other psychogenic reaction. Furthermore, a positive opinion as to the real nature of a "postconcussion syndrome" may depend upon knowledge as to whether or not a given patient's daily activities are consistent with the degree of disability implied by the subjective symptoms which have been alleged.

3. Certain focal lesions of the brain predispose to, if they do not actually cause, the appearance of epileptic attacks. Patients who sustain definite brain injury with clinical evidence of some form of persistent damage which is not well focalized may develop epileptic seizures. It is maintained by some workers in epilepsy that every epileptic possesses within him a gene which makes the convulsive reaction possible. The statistical evidence in favor of this theory is plausible and includes considering the argument by analogy, with respect to the physiological and biological similarity between epilepsy, migraine, and the more common allergic states. Assuming that there must be an inborn predisposition to the development of epileptic seizures, brain lesions might provide a precipitating basis.

In the ordinary epileptic, without any history of injury to the brain, one frequently finds both by history and examination various indications of an unstable vegetative nervous system which has been in existence before the first epileptic symptoms. As a rule, one cannot state the precipitating cause for epilepsy. When epileptic seizures follow a definite brain injury, one must search for the indications of a predisposition to epilepsy. The greater the amount of such predisposing evidence, the more possible it becomes for even a slight brain injury to precipitate actual epileptic seizures. The slighter the head injury, the more careful one should be in accepting such an injury as the cause for fits and the more need there is for making sure that such seizures had not occurred prior to the injury.

In brain injuries which cause focal damage in epileptogenic zones, there may be sufficient scar formation to cause a definite focus of irritation. In such cases, there is less dependence upon a predisposing inherited tendency to epilepsy. That is why one may point out in certain cases that epileptic seizures may well develop at a later date. Seizures which occur on the basis of localized brain damage are apt to be of focal Jacksonian type. In any event, the combination of focal brain injury and later Jacksonian epilepsy is pretty strong evidence of causal relationship.

If one is to predicate a direct relationship between brain injury and epileptic fits, through the intermediation of scar lesions, then the date of onset of seizures should conform with what is known as the duration or course of posttraumatic scar formation. The appearance of grand

mal or generalized epileptic seizures within a few days of an apparently inconsequential head injury should arouse one's suspicions that there is some other explanation for the fits than the injury itself. In persons who have sustained a severe focal injury to the brain, with the delayed development of what is called a porencephalic cyst, fits may not appear for months or even years after the injury.

At times, one is confronted with the problem of deciding whether a head injury is the result of a fit. It should be kept in mind that an epileptic always has his first attack and may have had good health previously as far as he could tell. A complete and accurate knowledge of the circumstances of an accident and the degree of violence exerted may enable one to state with practical certainty that an epileptic fit was responsible for the head injury. In such cases, later seizures are continuations of the process which caused the first fit, although permanent brain damage incurred in the first fit may perhaps have a modifying influence on the frequency and character of the later fits.

There is a point about the prognosis of individuals in whom head injury has apparently precipitated epileptiform seizures which do not have a demonstrable structural basis and which are accompanied by some of the somatic or constitutional evidence of an epileptic predisposition. I have been struck by the fact that seizures in such cases may cease within a year, whether or not any special treatment measures are used.

The clinical diagnosis of lesions in the brain due to injury may be very difficult. Clinical examination may have to be supplemented by special procedures such as encephalography. In interpreting encephalographic films, consideration must be given to previous illness or injury, clinical evidence of conditions which might be associated with structural cerebral defect prior to injury, the severity of the injury itself, and the time which has elapsed between the injury and the encephalographic examination. We do not have accurately established norms for encephalograms. Nevertheless, there are rough standards of normality. Not infrequently an encephalogram reveals findings, deviations from the average, which cannot be definitely related to either trauma or any other known life experience of a patient. Furthermore, a normal encephalogram does not preclude the presence of lesions which may be causing symptoms. Finally, an encephalogram may reveal very definite abnormalities of structure of long duration which apparently have not caused any symptoms whatever.

It is safe to follow the general rule that the more rapidly developing changes will manifest themselves by proportionately obvious symptomatology, and that if repeated encephalograms show progress in some abnormality, the cause is determined with greater certainty.

The interpretation of encephalograms is a matter requiring not

only skill in the doctor, but also a sufficient number of films taken and developed properly. Films must be stereoscopic.

RELATION OF HEAD INJURY TO PRE-EXISTING DISEASE

The problem of causal relationship between injuries to the brain and various pre-existing or independently accompanying diseases of the nervous system is limited to the problems of precipitation or aggravation. Again, it should be kept in mind that the term "aggravation" should be restricted to instances in which injury is demonstrated to have produced a temporary exacerbation, or else an acceleration or permanent alteration of the course of the pre-existing or independently accompanying disease.

Brain injury may have a precipitating or aggravating influence either through the production of lesions which have a demonstrable clinical effect or a sufficient degree of shock to disturb the systemic balance and permanently set into play conditions which have either been latent or clinically relatively inactive up to the time of the injury. In all cases, determination of causal relationship includes consideration of not only the lesions produced by trauma, but also the time factor between the trauma and production of symptoms of the pre-existing or independent disease.

1. *The apoplexies.*—Cerebral blood vessel lesions may occur on the basis of pre-existing disease of the blood vessels with or without abnormalities of blood pressure. There may or may not also be pre-existing heart disease which may be responsible for an apoplexy due to the effects of systemic shock upon the heart muscle function. A head injury which produces severe systemic shock may bring about a slowing of general circulation and a clogging or clotting of the blood in a cerebral vessel (thrombosis). If a cerebral injury is accompanied by a rise in blood pressure, nontraumatic hemorrhage from a brain vessel may result, if there is a background of blood vessel disease and elevated blood pressure.

Cerebral emboli reach the brain via the arteries ending in it. These vessels are the vertebral arteries which arise from the first part of the subclavian arteries, and the internal carotid arteries. Traumatized arteries leading to the brain may have inflammation of their walls and emboli may be broken off to lodge in the brain. Emboli may travel directly to the brain in cases of pulmonary disease and in certain types of valvular heart disease. Microscopic infective bacterial emboli may be carried to the brain in cases of septicemia.

Without any violence to the body from outside, sudden extreme or unaccustomed muscular effort may precipitate an apoplexy in a person with abnormal blood vessels. In general, it may be said that if physical trauma or adequate muscular effort precipitate an apoplexy, the symptoms of this apoplexy should appear either immediately or

within less than an hour. Injury associated with severe systemic shock justifies a longer time interval. Injury to the head which does not produce any definite alteration of consciousness or sharp pain and is not accompanied by any circumstances which would naturally cause a sudden severe change of blood pressure, is extremely unlikely to precipitate an apoplexy. Every 15 minutes delay between slight trauma or sudden physical effort and symptoms of an apoplexy makes any causal relationship less likely. In instances where precipitation of apoplexy is alleged to be the result of minor trauma to the head, or to result from a fall, it is either possible or likely that the "accident" arose out of the patient's inability to control his body as a first sign of an apoplexy. Unfortunately, one seldom is in a position to have accurate information as to exactly what has occurred. Long before such patients get to a neurologist they have prepared a story—sometimes quite honest—which implies that the accident was due to some environmental cause and not something going on within the patient's body or brain.

I have never seen or heard of a case in which an initial slight apoplexy, presumably precipitated by "accident," in which the injury was trivial, had any accelerating influence upon a pre-existing sclerotic disease of the blood vessels, nor do I believe that such a thing is medically possible. I have not seen but I have heard of seemingly acceptable cases in which serious brain injury precipitated or was accompanied by serious apoplexy with apparent acceleration of the pre-existing blood vessel disease. Liquefaction at the site of an apoplexy may predispose to later hemorrhage in the same region; such a late sequela should give clear cut symptoms. The difficulty in determining whether pre-existing blood vessel disease has its course accelerated by apoplexy lies in the fact that the rapidity with which such a constitutional defect progresses varies widely in cases without any complicating or coincidental trauma. Arteriosclerotic disease of the blood vessels with or without blood pressure abnormality depends for its rate of progress a great deal upon whether or not the patient has complicating organic disease of the heart, kidneys, or other organs, or some metabolic or debilitating disease such as diabetes, tuberculosis, cancer, systemic syphilis, or even obesity. In attempting to evaluate the possible aggravating influence of an accident in which the injury is complicated by apoplexy and accompanying systemic disease, the issue is complex and it is easy to see why equally competent physicians may disagree on the point of aggravation in a given case—especially if they are incompletely informed about relevant details.

2. *Syphilis of the nervous system.*—In the first place, it should be kept in mind that syphilis of the nervous system may occur in individuals who have no knowledge of exposure to the infection; secondly,

in perhaps 30 percent of the cases of syphilis of the central nervous system, the serology of the blood and spinal fluid may be negative. The mode of onset of syphilis of the nervous system may be gradual or abrupt. Finally, the clinical picture of syphilis of the nervous system is extremely varied, although the symptoms of the disease may be referable to only one portion of the nervous system.

In patients who show the physical signs of systemic or nervous system syphilis at the time of a head injury, it may be possible to determine whether the patient manifested any symptoms of the disease prior to the accident. Questioning may bring out evidence of the disease which the patient either paid no attention to or attributed to other causes—whether or not medical attention had been sought. It is thus obvious that a patient who has sustained a head injury should be submitted to an adequate general physical examination. When the clinical evidence of central nervous system syphilis is found, a neuropsychiatric examination should be arranged promptly. In my experience, it is common to find that the neurologist enters into the problem at a late date and not infrequently the delay contributes to difficulty in a determination of whether there is any aggravating causal relationship between the injury and the syphilis of the nervous system.

As a general principle it can be maintained that unless an injury is probably capable of producing or has actually produced clinical signs of lesions in the nervous system, there is no causal relationship between the injury and nervous system syphilis which is discovered later. Whether mere brief shock may precipitate or aggravate syphilis of the nervous system is highly speculative—almost an absurdity. Occasionally one sees a patient who develops symptoms of some form of neurosyphilis rather acutely after an intercurrent systemic infection. The effects of such an infection on the body's resistance to disease are really not comparable with, but are greater than, results of an injury which causes slight pain and does not confine the patient to bed because of serious shock. When a syphilitic disability is apparently precipitated by either an acute systemic illness or an injury in which there is ground for believing that the trauma caused actual lesions in the nervous system, the course of the syphilitic disability will vary in wide limits, just as is the case in patients who have no apparent precipitating cause.

When a head injury has produced definite lesions of the brain and there is pre-existing syphilis, especially of the nervous system, these lesions may be complicated by a syphilitic inflammatory reaction. Strauss has expressed considerable skepticism as to the possibility of paresis being precipitated by injury to the head. Other equally qualified neurologists who maintain an attitude of care, do hold that symptoms of cerebral syphilis may be precipitated by brain injury.

Of course, a person with cerebral or spinal cord syphilis may fall or be subject to an accident because of mental or physical handicap due to the disease. I have seen a few cases in which I was satisfied that a brain injury either precipitated or aggravated a pre-existing cerebral syphilis. I do not believe that a scalp laceration alone could have any effect. In such instances, the severity of the injury, the patient's known ability to work and get about before the injury and the time interval between the injury and the development of symptoms of cerebral syphilis all have to be weighed. Syphilis in parts of the nervous system or body not directly affected by traumatic lesions, will not be influenced by trauma, except through prolonged severe shock.

3. *Tumors of the brain.*—As in the case of syphilis, if a head injury has produced a lesion in the brain, it is conceivable that the pre-existing tumor may have been affected directly by some lesion which may apparently accelerate the progress of the tumor. Whether a head injury can actually cause a tumor cannot properly be determined by even a very carefully detailed study of a given patient. One should depend for judgment upon suitable statistical evidence. The incidence of the various forms of brain tumor in those who sustained head wounds during the Great War has been no greater or different from the incidence of brain tumors in people of the same age group who have not sustained head or brain injuries. This would indicate that trauma cannot cause a brain tumor and could have nothing more than an accelerating relationship to a pre-existing brain tumor. Since the life history of tumors of even one particular histological variety may vary considerably, the question as to whether in a given case trauma has acted as an accelerant should await a histological diagnosis. Without this aid, a claim of acceleration is on a highly speculative basis. The end result of a brain tumor, whether death, permanent partial disability, or even recovery, is not contingent upon what may appear to have precipitated or accelerated its course.

Cushing in his recent book on the meningeal tumors was impressed by what he regarded as evidence that they may be caused by trauma. One must take his opinion respectfully because of his vast experience. Nevertheless, Ewing's criterion cannot be disregarded—namely, that unless one can show that there was no tumor tissue present at the time of the injury, or that the injury produced lesions in the area where a tumor develops, it is improper to hold that single injury may cause a tumor to develop. The knowledge of a pathologist fully experienced in neoplastic disease will be more valuable than the opinion of the ordinary clinician.

4. *Acute infections of the nervous system.*—Infections due to pyogenic organisms which may enter into tissues damaged by trauma are obviously directly caused by the trauma. Fractures of the skull which

are compound and permit entry of bacteria from outside the body to the intracranial contents may result in some localized infection or a generalized meningitis. Fractures of the base of the skull which allow bacteria to penetrate in the sinuses or from the middle ear, may result in local or generalized infection of the brain and nervous system.

I have never seen a case of tuberculous meningitis, ordinarily a fatal disease, which had been precipitated by head injury. One could not deny, however, that a head injury producing an intracranial lesion might result in pre-existing tuberculous inflammation becoming active. Also, a patient with either active or arrested tuberculosis of the lungs or other organs subject to a direct trauma, might have the infection disseminated and as a result of spinal cord or brain involvement develop central nervous system tuberculosis. The time factor is important in any such instances.

Various so-called virus infections of the nervous system, of which epidemic encephalitis and infantile paralysis or anterior poliomyelitis are the most common, have their avenue of entry through the nasal passages and lymph channels between the base of the brain and the frontal, ethmoid, and sphenoid mucous membranes. It is not believed by any workers in virus diseases of the nervous system that the infection can enter the nervous system directly through trauma to the scalp or vault. The incubation period between entry of virus infections and clinical symptoms of the diseases varies from a few days to several weeks. If a head injury produces definite lesions during the invasion period or even after the virus infection has manifested itself clinically, the injury may have an aggravating influence. Whether it actually prolongs the disease or may result in death may be very difficult to determine. Seriously sick patients with encephalitis or poliomyelitis who are delirious may bang their heads against their beds. No one believes that such injuries have any effect upon the course of the disease. Again, when it comes to being definite about causal relationship between trauma as an aggravant of virus infections of the nervous system, one is confronted by the fact that statistical evidence must to some extent be depended upon. I do not know of any comprehensive statistics based upon well-described and properly classified clinical material.

5. *Degenerative and ill-defined diseases of the nervous system.*—

Paralysis agitans, multiple sclerosis, amyotrophic lateral sclerosis, subacute combined sclerosis, the myopathies, and cerebral arteriosclerosis represent the most frequent forms of disease in the classification, in which head injury may be alleged to have some influence.

None of these diseases are caused by trauma. Each has its own life history. Head injuries can have no permanent effect upon these conditions unless they happen themselves to affect the brain and the brain has been definitely damaged by trauma. Of the above-named

conditions, multiple sclerosis, paralysis agitans, and cerebral arterio-sclerosis naturally involve brain tissue. The other disorders are infrequently accompanied by any known lesions in the brain and therefore, unless such lesions occur, cannot be regarded as affected by an injury to the head.

6. *The intoxications.*—People with diabetes, occupational exposures to lead and certain other heavy metals, chronic alcoholism, or with the chronic organic residuals of carbon monoxide or other industrial gas poisoning, may sustain head injuries which may cause additional symptoms due to precipitation or aggravation of the pre-existing or predisposing conditions. Again, the course of such disability which is a combination of injury and systemic disorder, is apt to be determined by the combination of factors. Unless the head injury involves a definite and severe brain reaction, the part played by injury will be transient.

THE PSYCHONEUROSES AND MALINGERING

There is a prevalent assumption among the laity that a head injury is fraught with dire possibilities. If the skull has been fractured, there is fear that something serious has happened to the brain which may result in paralysis, serious mental defect, and all sorts of things. Even if there has been no fracture of the skull, the lay individual is fearful. His attention is apt to be focused upon the possible serious significance of even minor and transient subjective symptoms. Through what the patient thinks of his injury, therefore, may arise a psychoneurotic complication which may be much more serious and disabling than the actual physical injury itself. Of course, a head injury may have a significance unconnected with anxiety of the sort just described. The hypochondriacally inclined person who will over-react to any unpleasant experience and dwell upon it finds in a head injury a glorious opportunity for maintaining an attitude of invalidism, and transfers his anxieties and dissatisfaction about life in general to the injury, so that symptoms which the patient thinks belong to the injury are persisted in perhaps indefinitely. The individual who finds the struggle for existence hard, and who may conclude that the injury will militate against his return to employment, will be inclined to prolong symptoms and make no effort to use his body and mind unless some form of compulsion leads him to exercise his faculties and adopt a more optimistic and healthful attitude. The individual with pre-existing disease which has been disregarded or which has not actually been a handicap, may very readily utilize a head injury as a basis for concluding that the pre-existing disease has become serious and that he is therefore unable to maintain a useful existence. There are other varieties of psychoneurotic reaction to head injury, but I think that the ones mentioned

are the ones most frequently found. Such reactions would be accepted as sincere and genuine on the part of the patient, as long as he is unaware of the mental mechanisms at play.

Any factor which tends to confirm a patient in his honest belief will naturally serve to prolong such psychoneurotic reactions as may follow a head injury. In compensation cases, the continuance of disability benefits not infrequently serves to fix the patient's belief that he is permanently handicapped. After a period of months, such a belief may become so firmly rooted that nothing whatever can be done to dispel it. One has to depend upon unpredictable events which may be sudden or so important to the patient that he unconsciously decides to give up the invalidism and directs his future attitude and conduct in accordance with the meaning of the intercurrent "miracle." Some doctors may unwittingly implant in a patient's mind the ideas which lead to a chronic psychoneurotic reaction, and either unnecessary or unwise treatment serves to crystallize the patient's belief in his disability.

Crystallization of a genuine attitude of invalidism accounts for the instances in which discontinuance of disability benefit or closing a case on a nonscheduled lump sum settlement basis do not bring about improvement or cure.

As a general rule, the person of good intelligence who is well adjusted to life will not develop a persistent psychoneurotic reaction to a head injury. It is almost certain that a patient who goes to pieces about a slight head injury misrepresents the facts when he alleges that there was no source of tension, conflict or discontent prior to the injury. The psychoneurotic who wants to get well is usually amenable to psychiatric treatment—especially if it is begun promptly. One often sees the psychoneurotic who during a period of a year or more after an injury is subjected to what are really unnecessary consultations, examinations, and laboratory tests which, if indicated at all, should have been attended to at the time of the injury. In proportion to the degree of fixation of the neurotic attitude, these later consultations and laboratory examinations fail to have any value as far as the patient's state of mind is concerned.

Sometimes such genuine neurotic reactions may be complicated with the passage of time by the appearance of conscious insight on the part of the patient as to the nature of his condition. In such instances, if the patient continues to maintain his concatenation of symptoms, he is on the way to becoming or is a full-fledged malingerer. In any event, he can no longer be regarded as totally disabled and treatment measures are not only futile but a prostitution of the practice of medicine. It is in such cases that the money motive must be ended by a cessation of compensation.

A malingeringer is a somewhat different person. In the first place, the malingeringer is consciously attempting to deceive somebody else. If he is a stupid malingeringer, his lying is not only obvious but ridiculous in its extravagance, and he behaves in a normal manner when he believes he is not being observed. The inconsistency between the malingeringer's behavior and his allegations is definitely greater and more constant than the inconsistencies which at times exist in the genuine neurotic whose attention may be temporarily distracted from his symptoms and who during such a period behaves as though he is not dominated by them. The smart malingeringer may be shrewd enough to seize upon one or two subjective complaints which cannot be proved or disproved by examination or observation. Another and rather unusual type of malingeringer is the individual who selects some one symptom such as paralysis of a portion of the body and for the duration of his "campaign" carefully avoids use of the allegedly paralyzed member. Differentiation between an hysterical paralysis and a fraudulently simulated paralysis of this sort may be very difficult. In the few such cases I have ever seen, the malingeringer was identified as such by the obviously conscious lying about the circumstances of his injury, the occurrence of various symptoms following it, the fabrication as to treatment given, and what was allegedly stated to him by physicians caring for him. In one instance fraud was apparent by the evidence which showed that a period of several weeks went by between the accident and the development of a "paralyzed" arm coincidentally with the retaining of an attorney.

It is widely held that the malingeringer does not have a normal personality. His pre-injury record may show the same traits and reactions which predispose to a genuine psychoneurotic reaction to injury. Therefore, differentiation is a matter of marshalling the evidence which determines whether or not the injured person is consciously motivated in his behavior and complaints.

INJURIES TO THE BACK

The spinal cord may be affected by mechanical force or trauma to the back, as a result either of force applied from outside to the same level as a lesion in the spinal cord resulting therefrom, or at times by indirectly transmitted violence. The majority of instances in which the spinal cord is affected occur in conjunction with direct damage to the vertebral column or its ligaments.

THE IMMEDIATE EFFECTS OF INJURY

1. *With vertebral injury.*—In association with vertebral injury there may be concussion or compression of the spinal cord. Concussion may be very slight and accompanied by very little in the way of

symptoms of which the patient is aware, and in such instances there is practically always spontaneous recovery. When a concussion is severe enough to abolish function at the level of the cord affected, the clinical picture is identical with that of crushing of the spinal cord or any other lesion in which there is abolition of function. However, in cases of severe spinal cord concussion there is a tendency to recovery. Residual defect or impairment of function, if present at all, is demonstrable objectively at least by abnormalities of reflexes.

Serious compression or destruction of the spinal cord produces its maximum clinical effects immediately. The same is true of a hemorrhage within the spinal cord.

In neck injuries, there may be a sudden stretching of the spinal cord, with not only damage to the nerve roots, but at times hemorrhage within the substance of the spinal cord (hematomyelia).

Indirect violence to the spinal column which produces a crushing effect may result in damage only to an intervertebral disc. The immediate effects upon the spinal cord are very often restricted to nerve root symptoms and signs. Early injury to an intervertebral disc may not be demonstrable by the x-ray.

2. *Without vertebral injury.*—The effects on the spinal cord, except for compressive lesions, are the same as those which occur with demonstrable vertebral damage.

Penetrating wounds, of course, offer a special problem. Stabbing and gunshot injuries are the most common varieties. I have seen one case of electrical burn in which the spinal cord was "cooked" at the level where there was contact between high voltage current wire and the skin. Ordinarily, electric shock will not cause a focal destructive lesion.

DELAYED OR LATE INVOLVEMENT OF THE SPINAL CORD

When there is chronic or progressive deformity of the vertebral column at the site of damaged vertebrae, or in instances where there is a traumatic herniation of one or more intervertebral discs, delayed spinal cord compression or other involvement may occur.

If there has been only a slight concussion or transient compression syndrome without vertebral damage and if there has been an apparent complete recovery of function, symptoms and signs of disease in the same region or elsewhere in the spinal cord appearing at a later date are not attributable to the injury. There are rare instances of apparently mild localized damage to the spinal cord in which recovery is not complete and because of nutritional defects there may be a progressive atrophy or softening of the spinal cord at the site of the original injury. Also, certain types of lesions may result in adhesions between the spinal cord and the surrounding meninges and the scars of such lesions may apparently be progressive in their influence and

cause a delayed but increasing impairment of function at the level of the original injury.

RELATION TO PRE-EXISTING OR ACCOMPANYING DISEASE OF THE SPINAL CORD

Unless the back injury has caused vertebral damage or definite lesions in the spinal cord, it can neither precipitate nor aggravate pre-existing disease of other origin.

A patient who has syringomyelia, has a cystic dilatation of the central portion of the spinal cord which is perhaps especially vulnerable in reaction to violence to the back at the same level. Syringomyelia cavities occur usually in the cervical and lumbar enlargements of the spinal cord and trauma directed to these regions may aggravate a pre-existing syringomyelia, with the production of a superimposed hematomyelia.

Tuberculous disease of the spine may be made manifest by a slight degree of trauma which causes an acute collapse of the diseased vertebrae with resulting spinal cord compression.

Certain metabolic diseases affecting a vertebra may predispose to a disproportionate effect from even a slight trauma such as a strain or suddenly assumed unusual posture. Vertebrae which are involved by metastatic cancers or other malignant tumor may be collapsed, or have the local tumor growth rate accelerated, by trauma directly affecting the diseased vertebrae, with consequent spinal cord compression or invasion by tumor.

Pre-existing tumor of the spinal cord or nerve roots may have its position so shifted by trauma that there will be an acute onset of a compression syndrome. This is a not rare sequel to diagnostic lumbar puncture for syndromes of cord compression and when it occurs the physician regards the complication as very helpful in establishing the diagnosis and enabling prompt surgical treatment. In such instances, the trauma is only a precipitant and does not have any aggravating effect upon the course of the tumor.

In cases of virus disease affecting the spinal cord, trauma can have no influence unless the trauma itself affects the spinal cord. The incidence of acute virus infections of the nervous system limited to the spinal cord is largely restricted to infantile paralysis. The likelihood of injury to the spinal cord occurring during the invasion period of such a disease is extremely small. Even if one were to run across such a case, the course of the disease would have to be obviously and immediately influenced by the trauma if there were to be any basis for aggravation. If a patient has recovered from infantile paralysis, even though the infection may have permanently damaged the nervous system, the virus is dead and cannot be reactivated by trauma. Therefore, injury to the spinal cord occurring after recovery from such an infection cannot aggravate disability which has already

reached its maximum, and any new disability is the product solely of the injury.

Epidemic encephalitis is a disease the virus of which may remain active indefinitely and in the unusual instances of this disease in which the spinal cord itself is affected, it is conceivable that adequate trauma may have an aggravating influence.

Patients with multiple sclerosis, combined sclerosis, myopathies, syringomyelia or other chronic degenerative or progressive diseases of the spinal cord show objective signs of these conditions. If such signs are found at the time of an injury to the back, it is usually readily determined that some or all of the signs must have preceded injury. Unless a back injury produces additional lesions causing an added independent disability, or is proved after an adequate period of observation to have actually altered the course of the previous disease, there is no causal relationship.

With respect to multiple sclerosis, the disease is one in which spontaneous transient exacerbations are characteristic and may in some cases be precipitated by all sorts of conditions which affect the body as a whole. Such intercurrent illnesses as tonsillitis, grippe, and appendicitis have been followed by transient exacerbations of the disease. A back injury which is accompanied by severe and persistent pain, but which has not affected the spinal cord, may serve to upset the systemic equilibrium and bring on an exacerbation of the multiple sclerosis.

I have never seen a patient with any form of myopathic disease whose disability had been affected by trauma. Many of these patients are subject to falls because of their handicap, but I have yet to know of any influence of such falls upon the pre-existing disease.

Syphilis of the spinal cord may be aggravated by back injury affecting the spinal cord. The only cases seen by me in which I felt this was true were in individuals who promptly developed localized level lesions of the spinal cord.

It is theoretically possible for a sufferer from tabes or syphilitic lateral sclerosis to have spinal cord damage from injury limit its effects to an acceleration of the pre-existing syphilitic disease, without evidence of any definitely localized syphilitic inflammation due solely to trauma.

THE PSYCHOSES, PSYCHONEUROSES AND MALINGERING

The principles discussed in connection with the mental reaction of a patient to head injury apply also to instances of back injury.

A considerable proportion of patients who have sustained back injuries are both orthopedic and neurological problems, and many of these individuals really are psychiatric problems in which the patient's attitude is the basic reason for alleged disability.

INJURY TO THE PERIPHERAL NERVES

The cerebral or cranial nerves may be affected by trauma to the skull or brain. The roots of the spinal nerves may be affected by trauma to the back or the vertebral column. The more peripheral portions of the cerebral and spinal nerves may be affected by injury to the surrounding soft parts, and in the case of spinal nerves by injury to bones in proximity to the nerve trunks.

IMMEDIATE EFFECTS

In trauma by penetrating wound, compression, or by stretching, if nerves are to be affected at all, there is almost always prompt clinical evidence of their involvement, characterized by pain and sensory loss in the area supplied by a nerve, and if the nerve carries motor fibers, some degree of loss of power and wasting of muscles supplied by the nerve. The cerebral and spinal nerves seldom offer any problem in causal relationship.

DELAYED EFFECTS OF NERVE INJURY

Compression lesions due to soft-tissue scar or bone deformity may be delayed and symptoms not evident for months or years after the time of injury. Scalp lacerations may result in scars which produce compression and irritation effects upon nerves. The head pain resulting therefrom must be differentiated from the head pain due to skull or meningeal lesions as well as the head pain which may be a sequel of concussion or other brain damage.

RELATION TO PRE-EXISTING DISEASE

As for the cerebral nerves, trigeminal neuralgia, deafness and labyrinth disease may present difficulties. The principles in determining causal relationship are similar to those which hold when trauma has been directed to the brain or the spinal cord.

Various forms of localized inflammation of nerve roots and single peripheral nerve trunks may be present at the time a patient is injured, but unless the previously affected nerve trunks or roots show evidence of direct added injury, there is no causal relationship.

Chronic alcoholics, diabetics, or those with occupational or other abnormal exposure to lead, seem to be predisposed to develop signs of nerve trunk disease, when there has been direct trauma of even slight degree.

Sometimes the peripheral nerve syndromes associated with intervertebral disc lesions may represent pre-existing intervertebral disc abnormality which has been aggravated by trauma. Patients with structural weaknesses in the back or developmental anomalies which

include "hypertrophied" spinal ligaments or congenital bands or adhesions in the lumbosacral region, may develop peripheral nerve syndromes with pain in the back and lower extremities, due either to adequate single trauma or to repeated slight traumata or strains.

People with rudimentary or fully developed cervical ribs or compressing fibrous bands are predisposed to symptoms referable to the eighth cervical and first thoracic nerve roots.

Mechanical trauma to one or two nerve roots or peripheral trunks will not have any relationship to systemic disease or disease of organs elsewhere in the body not supplied by the affected roots or nerves.

Multiple neuritis is a manifestation of systemic infection or intoxication. It is not caused by mechanical trauma to nerve roots or peripheral trunks. The various intrinsic diseases of the spinal cord and brain are neither precipitated nor aggravated by injuries to the peripheral portion of the nervous system.

PSYCHOSES, PSYCHONEUROSES AND MALINGERING

No organic psychoses in which there is structural disease in the brain can be causally connected with an injury limited to the peripheral portion of the nervous system. The constitutional forms of psychoses and psychoneuroses would have causal relation only through the fact that the disability connected with the injury itself furnishes or is linked with an unconscious conflict. Whether the injury has only a precipitating influence will depend upon the particular conflicts and maladjustments of the given patient. In my experience, patients with peripheral nerve injuries with psychoneurotic complications are rare, and when the complication occurs it has been connected with either difficulty in adapting to a necessary change of occupation, or the fact that the injury is responsible for some persistent cosmetic or other physical handicap.

Malingers rarely simulate a peripheral nerve paralytic syndrome. They seem to confine themselves to complaints of upper arm and shoulder pain with "weakness" or restricted movement, or to variations of the "sciatic" syndrome. Such individuals may puzzle the best doctors, but complete examination, and intelligent observation will reveal the truth.

VASOMOTOR DISORDERS

Localized vasomotor disorders may result from direct trauma to cerebral or spinal nerve trunks. There are certain systemic conditions in which circulatory defect and tissue metabolism impairment occur. The true vasomotor neuroses are characterized by defect of innervation of blood vessels, and some syndromes have a progressive proliferation of the endothelium or inner lining of the vessel walls and decreased blood flow. Often, all four extremities show evidence of involvement.

In arteriosclerosis, and in diabetes, tissue metabolism is not normal. If such predisposing systemic conditions are complicated by either peripheral nerve trunk or other soft tissue injury, vasomotor and tissue nutritional defects may be aggravated. Varicose and arteriosclerotic ulcers may be aggravated; arteriosclerotic and diabetic gangrene may result.

Direct injury to an artery with permanent decrease of blood supply may result in vasomotor or other trophic disorders in tissues supplied by the affected artery.

There is in my opinion no medical basis for regarding an injury to one part of the body as able to alter the course of a pre-existing generalized systemic defect, of the sort mentioned above, unless in the particular case the local effects are of such degree and character that the body as a whole obviously suffers.

THE PSYCHOSES, PSYCHONEUROSES, AND MALINGERING

RELATION TO INJURIES NOT INVOLVING THE NERVOUS SYSTEM DIRECTLY

Injuries not directly affecting the nervous system may indirectly have an effect upon it either through severe shock or persistent suffering or debilitation. In this manner, certain types of psychoses may be precipitated—those of the toxic infectious group as well as the so-called constitutional psychoses.

Psychoneurotic reactions and even constitutional psychotic states may be the result of the meaning to the patient of an injury to some part of the body. Injuries affecting the heart and the genitals, or causing symptoms referable to heart function, seem to be rather commonly associated with some type of psychogenic reaction.

Operations for hernia have been known to apparently precipitate severe psychoneuroses and occasionally even dementia praecox or a manic-depressive episode.

In order to accurately and fairly determine the degree to which accident or illness is a causal factor, one must make a psychiatric survey of the patient and give special attention to predisposing defects of personality. In cases without medico-legal issues, many of the various so-called precipitating causes of psychoneuroses and psychoses are regarded as having little or no influence whatever on the course of the mental disorders. Often it can be satisfactorily demonstrated that the patient was already suffering from his personality disorder and that the so-called precipitating factor had no actual causal connection at all. It was the patient's existing mental disorder which caused him to react abnormally to some inconsequential incident, minor intercurrent illness, or physical injury. It is therefore important to promptly recognize and inquire into psychoneurotic and psychotic symptoms developing in an injured person.

In such cases, the mere *post hoc ergo propter hoc* standard of causal relationship is utterly unscientific. The attempt to treat a patient on the ground that an actually irrelevant incident is the cause of his disorder would be disastrous.

Conscious fabrication, exaggeration of symptoms, and persistence in complaints which have no physical basis—usually rather obviously related to the desire for money—may occur in individuals who have had any sort of injury.

BIZARRE PAIN IN THE EXTREMITIES¹

By Commander Rodney F. Atsatt, Medical Corps, United States Naval Reserve

Three years ago a paper was presented, before this assembly, dealing with the production of referred pain and in fact actual visceral disturbances, through the mechanical irritation of nerve roots and plexuses as they lie on or pass through paravertebral muscles irritated by a scoliosis. It would seem now that some observations of referred pain in the extremities might be worth bringing to your attention.

Referred pains, for that is what most of these bizarre painful affections are, may be taken by definition to be due to secondary neuritis. One says secondary neuritis advisedly for there are few common, primary neuritides. The notable example of a confusing primary neuritis is of course herpes zoster or shingles. This inflammation must always be considered in diagnosis, though it is rare on the extremities.

Neuritis may be secondarily caused by a number of pathological states. The exact mechanism of spread of the inflammation is not always clear but evidence of it is seen in the nerve trunk tenderness and muscle atrophy as well as in the pain and paresthesias which accompany the usual secondary neuritis. It is not to these frank manifestations that I wish to call your attention but to those cases where the local peripheral symptoms are the important presenting indications and where the primary irritation is obscure.

In the upper extremity let us first consider the type of pain that accompanies cervical lesions. The first and most common lesions to be considered are cervical arthritis and myositis which may give pain in the whole or any part of the upper extremity. This pain may be accompanied by tingling and numbness in thumb and fingers with very slight local manifestation of discomfort in the neck.

Thus with unusual pain down one or both arms, often accompanied by paresthesia, our first search for cause should be in the cervical spine and associated muscles. Tumor of the spine or cord or posterior triangle of the neck must also be considered but here we would usually

¹ From the Santa Barbara Clinic. Read before the Western Academy of Orthopaedic Surgeons, November 1939.

find collateral symptoms which might indicate the diagnosis. Cervical rib and aneurysm of the subclavian artery are some of the rarer primary lesions, and, of course, left arm pain accompanied by cardiac discomfort is always suggestive of angina pectoris.

But there are less obvious causes for this neuritis. A subacromial or subdeltoid bursitis may be first suspected when a patient comes in complaining of pain in the lower forearm, wrist, dorsum of the hand, or extending down into the third and fourth fingers. This distribution is peculiar, being neither purely segmental nor by trunks, but nevertheless repeated so often we must accept its validity. It probably is due to the establishment of an irritable focus of McKenzie in the cord initiated by afferent impulses from the bursitis.

In bicipital bursitis, or more properly, bicipital tenosynovitis, with local tenderness over the sheath of the long head of the biceps, the first symptoms may again be referred pain down forearm, wrist, hand, and fingers, often including the thumb. Very often cases are seen with no recognized symptoms in the shoulder and with pain in this distribution, unaccompanied by shoulder tenderness, and yet one of our early considerations is that of bursitis, because of the pattern of radiation pain which is presented.

In this connection it may also be mentioned that irritation of the upper bicipital tendon is occasionally reflected in pain and tenderness in the insertion of the biceps into the radial tuberosity. This is of course a direct myositis or fascitis of the muscle or sheath and needs no interposed neural factor for explanation.

May I turn aside a moment to emphasize a statement just made. Pain especially of the flashy, stabbing type, unaccompanied by local tenderness should always make one suspicious that it is referred rather than local in origin. The sprain, the fracture, the bruise, the hematoma, and the osteomyelitis, while they may exhibit intense pain, all have as concomitants, local tenderness and often swelling and change in color. Referred pain has none of these. It stands alone like a signal beacon by which the shrewd observer may profit.

Returning to the upper extremity again we see tennis elbow causing referred pain to the wrist, but here the mechanism is similar to that just described in the bicipital muscle reaction, inasmuch as the inflammation at the epicondyle of the humerus spreads either by way of the muscle substance or by way of the muscle sheath to the insertion at the base of the first metacarpal. Thus, again one finds referred pain in the wrist attributable directly to a pathological lesion higher in the arm.

Occupational neuroses also play a part in this picture of bizarre pain in the upper extremity. Writer's cramp, sewer's cramp, etc., reflect a muscle spasm caused by an oft repeated movement of the

forearm, wrist or hand, and while not truly referred pains they are certainly bizarre in origin.

Then, too, one must not forget the anemias as causing referred pains. The neuritis of primary anemia is too well known to be more than mentioned, but pain in the arms and legs also often accompanies a true secondary anemia.

In the lower extremity the causes of pain may be many. Sciatica is, of course, the general term that covers several conditions. For a detailed discussion of this syndrome, one is referred to the first of a series of articles dealing with sacroarthrogenetic telalgia by Pitkin and Pheasant.²

Referred pain here may arise from arthritis of the lumbar spine, paravertebral muscle spasm, involvement of the lumbosacral joints and involvement of the sacro-iliac joints. Of course, from a segmental standpoint one would expect these widely separated origins to produce various localizations of pain. Here again it is well to emphasize that pain unaccompanied by *tener*, *rubor*, and *tumor* should make one ponder seriously as to its origin.

In opening this discussion of lower extremity pain, it is hardly necessary to review the segmental distribution of the lumbar and sacral nerves, as the many authorities admit the possibility of participation of several different structures in the origin of these referred pains. In general, we may say that the femoral area is supplied by the lumbar nerves and when a patient complains of pain in this region uncomplicated by tenderness or loss of motion in the hip, one instinctively looks for trouble in the lumbar spine. Similarly, when discomfort is found in the gluteal region we think of strain or other pathology in the upper sacral joints, either lumbosacral or sacro-iliac. Pain down the back of the thigh is, of course, typical sciatica and is often accompanied by true trunk tenderness. In accordance with one's teaching, one will look for the initial lesion in the sacro-iliac joint or the lumbosacral joint, or in the unbiased mind, in both.

Also when a man is seen complaining of pain in the outer aspect of his knee joint (unaccompanied by tenderness) one's first thought should be of a process in the sacro-iliac joint and not of an external semilunar cartilage injury. Again, when a person complains of pain in the outer aspect of the leg and reports that he "must have bumped it," be sure there is local tenderness and some other evidence of local injury before you send him home for hot compresses to the leg; otherwise some colleague will finally relieve him by strapping his back! Most authorities agree that the outer aspect of the leg is supplied by lumbar V, and sacral I and II. As indicated above, to some observers this distribution implicates the lumbosacral joint

² Pitkin, H. C. and Pheasant, H. C.: Sacroarthrogenetic telalgia, Jour. Bone and Joint Surg., 18: 111, January 1936.

with a foraminal nerve irritation, a true radiculitis. To other observers it indicates a neuritis from association with an inflamed and irritated pyriformis and thus it is referable to the sacro-iliac joint. To still others this telalgia originates in the posterior sacro-iliac and sacro-ischial ligaments. Suffice it to say that to whatever school of thought one gives allegiance, to the wise clinician, uncomplicated pain in the outer side of the leg and down over the dorsum of the foot indicates some form of therapy for the low back.

How many times one has complaints of pain in the ankle and foot, especially the outer side and dorsum which is still the lumbar V, sacral I, II distribution. It often takes a lot of persuasion to convince a patient whose whole mind is centered upon a painful foot or great toe to allow one to make an examination of the back. It should be done, however.

Now for some of the less frequent causes of referred pain in the lower extremity unassociated with local signs and symptoms. Pelvic inflammation, tumor mass or retroverted uterus may give referred pain. Disease of the bony spinal canal, meninges or cord will also naturally affect the leg. It is quite unnecessary to discuss in detail obturator neuralgia in the knee region from hip disease, multiple neuritis from avitaminosis or toxemia, and the lightening pains of tabes. Intermittent claudication from thrombo-angitis obliterans gives pain in the lower extremity with but few associated signs. The early pain of erythromelalgia may be confusing since it may precede the typical flushing and increased heat. Occasionally one sees referred pain from bursae over the ischial tuberosities (weaver's bottom) and often the discomfort from trochanteric bursae radiates down the outer side of the thighs.

We cannot close this paper on bizarre pains in the extremities without mentioning two other conditions which, while they lead us away from our precept of referred pain without the local symptoms of tenderness, swelling and redness, are nevertheless somewhat odd in their origin. The first of these conditions is painful heel. This of course may arise from a multitude of causes. Periostitis of the os calcis from constant jarring, diffuse cornification of the skin, chillblain and fibrositis of the soft tissues may be mentioned as general factors. Specific causes such as injury, tuberculosis, osteomyelitis or foreign body may also be mentioned, but these are obvious to an intelligent clinician. The type of discomfort that may be baffling is that tenderness and pain ringing the base of the calcis with an especially tender and painful spot in the center of the heel. When an x-ray is taken and a magnificent spur is seen on the under side of the os calcis the problem is solved—until one glances in passing at the roentgenogram of the other heel and sees that the spur there is just as large or larger. What

makes the painful difference? The tender one presents evidence of a periosteal reaction due to the pull of a stretched plantar fascia—put on stretch by a lengthening and depressed longitudinal arch. All the cupped heels in the world will not give comfort unless the longitudinal arch is supported.

Though this may seem very elementary, there is one more observation which must be added. Always think of gout. Many a great toe is treated for an inflamed bunion when in reality a metabolic imbalance is the causative factor—and a dietary regime would be a curative agent.

Referred pains are like the old-fashioned shell game. The urchin with his eyes glued upon the peas watches them disappear and reappear under the shells and remarks, "The blamed things just ain't where they orter be."

THERAPEUTIC ASPECTS OF THE PSYCHONEUROSES *

By Annette C. Washburne, M. D., F. A. C. P.¹

The psychoneurotics like the poor are always with us. They can be counted on to fill the office when all the "interesting" cases have disappeared. Their numerous and recurring complaints and their unstable autonomic systems are almost sure guarantees for numerous visits and night calls. Yet all this the average physician would cheerfully overlook were it not for the troublesome question of therapy. One hears repeated in papers and at meetings the unvaried question "but what can you do for them?" Perhaps the whole problem would be less dismal if one approached it with certain rather definite things in mind.

(1) One may assume, for purposes of clarity, that the psychoneuroses represent maladjustments of the individual to his environment.

(2) One must concede that an isolated psychoneurotic episode does not constitute a psychoneurosis in the general sense of the term. A "normal" person (that is one who is symptomless) may under sufficient provocation react in a neurotic fashion. After the causative factors have either been removed, or the patient has adjusted himself to them, he once again becomes "normal." These reactions, while significant, are part of the general pattern of living. They may be anticipated as is the common cold and have, quite possibly, the same potentialities for becoming more serious. The "normal" person undergoing a neurotic reaction presents a relatively simple therapeutic problem in that there is something to work with, and such reactions are usually of short duration.

(3) There remains the psychoneurotic reaction in the inadequate individual. The reaction itself may present many of the symptoms

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seen in the normal person's neurosis but there will be certain well defined variations: (a) The symptoms will tend to be more exaggerated, (b) to recur with far greater frequency, and (c) to arise in response to minor provocations. In this case the therapeutic problem will be difficult since there is little to work with and because of the longer duration of the neurosis and its chance for recurrence. If these two points, however, are kept in mind the physician approaches the case with the knowledge that treatment will, of necessity, be extended over a period of time. He may anticipate temporary remissions but he must expect frequent exacerbations. To refuse to treat a psychoneurotic on the twentieth occasion just because "we settled all that before" is equivalent to refusing morphine to a carcinoma case because she had an injection 3 hours previously. The gaining and the maintenance of this point of view are of extreme importance. It will be manifested in the physician's approach to the patient. As a result he is more tolerant, less brusque, more philosophical in his attitude. Correspondingly, his therapy will be more effective.

(4) Finally, as to the therapy itself: It is quite simple to formulate generalities such as "get the story—desensitize—reeducate." The difficulty comes with the high degree of individualization required. Certain points, however, appear clear. The majority of psychoneurotics have numerous subjective somatic complaints—palpitation, tremors, anorexia, nausea, shooting pains, tense muscles, insomnia, *et cetera*. Since these phenomena are actually present the individual, highly aware of the symptoms, is inclined to use them as stepping stones for fear projection. Thus ideas of heart disease, central nervous system involvement, peptic ulcers, carcinoma, and so forth, are only too frequently postulated. Many physicians unfortunately do little to decrease these fears since, regarding them as neurotic manifestations, they either fail to examine the patient at all or do so in a haphazard manner. That either of these procedures may be highly dangerous is proved by the frequency with which organic pathology is found in the neurotic individual.² A careful history and painstaking physical and neurological examination are of first importance. Whether or not there is a disease process is, for our purpose, irrelevant. The patient begins "right" with the doctor. Confidence and rapport are established. If the examination is followed by necessary laboratory work this relationship will be further enhanced. In this regard it would seem that an electrocardiogram or an orthodioscopic examination would be amply justified in a cardiac neurosis. Following such procedures the physician can say, with greater assurance as far as the patient is concerned, that no organic disease is present. He can then explain the mechanism of the "nervous heart" so that the patient will

² Comroe, B. I.: Follow-up study of 100 patients diagnosed "neurosis." *J. Nerv. & Ment. Dis.*, 83: 679-684, June 1936.

understand. (That such explanations will have to be patiently repeated many times is an accepted fact.)

We have been interested in the frequency with which fatigue is complained of by psychoneurotics. Quite possibly tension and fatigue are two aspects of an X-factor. Since, in many of these patients, the fatigue appears worse when the patient is fasting or when considerable time has elapsed after the last meal, sugar tolerance tests were taken on a representative group. (These findings will be reported completely at a later date.) With few exceptions the curves were found to be relatively low. Patients of this type almost uniformly felt better when given supplementary carbohydrate feedings and, where it was not contraindicated, small doses (25 mg.) of ephedrine once to twice daily. We are aware that there are many physicians who would heartily disapprove of any such approach. Their objections are usually based on: (a) "Waste" of money, (b) unscientific medicine, and (c) unwarranted pampering. None of these objections appear valid to us if we grant that the psychoneurotic is a sick person, that he has certain accompanying physiological disturbances, and that we must begin treatment **somewhere**. Psychologically, doing **something** which gives **rapid**, though possibly temporary, relief of some phase of the picture appears to us justified. Especially does this seem the case when one realizes that what the physician will be able to do for the patient from there on will largely depend upon the confidence and reassurance the physician is able to give. By some such simple physiological procedure or by the use of the bag as recommended by Kerr ³ the first step is taken.

Following this, the problem arises of listening to the whole story. Time and patience are essential. It seems of utmost importance that the physician should never appear surprised, shocked or, what is worse, already cognizant of what is being told. Thus a patient who was exceedingly worried and sensitive about frequent masturbation consulted a doctor. After much embarrassment the patient disclosed his difficulty. The physician assumed a knowing air and said he had guessed it from the first, the patient left his office convinced that his difficulty was perceptible to anyone who talked with him.

Nearly all psychoneurotics will manifest uncertainty, fear, and depression and many will show hostility, suspicion, and excitement. Since these symptoms have been called forth in response to a situation which the patient interprets as threatening, and since the symptoms themselves are outward manifestations of inner conflict, effective therapy should consider both aspects. In other words the symptoms must be alleviated and the conflict adjusted as far as possible. In the chronic psychoneurotic the latter consideration constitutes the most

³ Kerr, Wm. J., Gliebe, Soley and Shock: Treatment of anxiety states, J. A. M. A. 113: 637-641, August 1939.

difficult part of the problem. Because of the frequency of poor heredito-constitutional factors and the added complication of psychosomatic deficiencies, conflicts in these patients are seldom settled for long. It would seem that in such cases, where one is dealing with dubious foundations, the best results will often be obtained by relief of symptoms, and by attempting to develop within the patient a more adequate design for living. In this latter respect one would consider those factors such as an irritating environment, loss of work, and so forth, which are capable of being removed or changed. The unchangeable factors such as body deformity and deep rooted personality traits must be faced and accepted in the manner best for the patient. Many people keep themselves in a state of chronic futility by attempting to break through immovable walls or to achieve impossible goals. The psychoneurotic suffers by competition with the "normal." If his limitations can be demarcated and he can be persuaded to live within their boundaries, better adjustment will ensue. Obviously such an achievement requires much tactful handling. One cannot, we feel, say as one physician advises, "You're a constitutionally inferior person so don't expect much." One can indicate, however, that no general standard of accomplishment is feasible for all and that for each person the honest recognition of what he can do is essential. To work out goals possible of some achievement is vital. Here the conscientious physician may have to assume the temporary role of social worker, defender, counselor, and planner. As soon, however, as the patient manifests capabilities of being able to do something, the physician gradually extricates himself. The patient is encouraged to go ahead with what he can do. If he feels physically better and psychologically safer he may progress for some little time and the inevitable return be delayed. It appears to us that successful treatment of the chronic psychoneurotic symptoms should be judged by the length of remissions rather than by their removal. The optimistic belief that in most chronic cases these symptoms can be eliminated seems to us evidence that the physician himself is challenging an immovable wall. Lastly, the following drugs have proved most useful for the reduction of some of the symptoms mentioned above:

Symptom	Drug	Dose
Fear.....	} Phenobarbital.....	$\frac{1}{4}$ - $\frac{1}{2}$ gr. t. i. d., a. c.
Tension.....		
Excitement.....		
Fatigue.....		
	Ephedrine (Supplementary carbohydrate feedings.*).	25 mg. daily to b. i. d.
Insomnia.....	Nembutal.....	1½ gr.
Depression.....	Benzedrine sulphate (cautiously as a trial only)*	5-10 mg. daily.

* Blood pressure check required at regular intervals.

CONCLUSION

It should be emphasized that if this discussion is criticized, as it justifiably may be, on the basis that it is a superficial oil pouring on troubled waters and that it does not penetrate sufficiently into the lower strata of conflict, psychomechanisms, *et cetera*, it can only be reiterated that from a practical point of view deeper therapy appears to us of questionable value in chronic neurotics. Unless the person possesses a reserve upon which one may count for more lasting adjustment, a complete "house cleaning" is of dubious benefit—after all one must have some furniture to rearrange later on.

HELIUM

By Lieutenant Commander G. W. Shilling, Medical Corps, United States Navy

Because of the present wide use of helium-oxygen mixtures, not only in deep-sea diving but also in the treatment of medical conditions, it seems worth while to review the literature on helium and to present some of the more interesting aspects of its discovery, properties and uses.

HISTORY

Lockyear, the brilliant English astrophysicist, in studying the solar spectrum in 1868 discovered a yellow line which, after exhaustive studies, he determined to be due to some new gas. This gas he called *helium* from the Greek word *helios* (sun). By peculiar coincidence, the French astronomer Janssen found the identical line, and their reports arrived at the French Academy of Medicine within a few minutes of each other. Ten years later Father Angelo Secchi verified their findings. In the year 1895 Ramsey discovered helium in the mineral cleveite and was thus the first to recognize it as a constituent of the earth's substance. Later it was found in other minerals and these furnished the first sources from which it was recovered. It was obtained in England by heating manazite sand imported from India but the amount recovered in this way was so small and the costs were so high that it could only be used for limited scientific investigation.

In Dexter, Kans., in 1903 a gas well was drilled which in spite of a good flow was a great disappointment because it was found to be non-inflammable and therefore worthless as a fuel. A sample of this worthless gas was sent to the University of Kansas where it was found to contain 1.84 percent helium, which was at that time costing about \$2,500 per cubic foot to obtain. Other natural gases were found to contain traces, and with the World War the Bureau of Mines¹ commenced, and has since continued to maintain, a comprehensive survey

¹ Stewart, A. About Helium. U. S. Bu. Mines information Cir. No. 6745; September 1933.

of the gas-producing areas of the United States for helium for Government purposes. At the present time, the main source of Government supply is the Amarillo field in Texas.

PROPERTIES

Helium is one of the rare gases. It is colorless, odorless, tasteless, and physiologically and chemically inert. It has a density one-seventh that of nitrogen and one-eighth that of oxygen, the molecular weights of the three gases being: Helium 4, nitrogen 28, and oxygen 32.

The diffusibility of a gas is in proportion to the square root of its density, so the amount of pressure required to move a mixture of 80 percent helium and 20 percent oxygen is just about half that required to move air. Not only less pressure and therefore less effort is required, but greater speed is obtained in the movement of helium through semipermeable membranes and narrow orifices.

The weight of helium under standard conditions is only 10.54 pounds per 1,000 cubic feet, while that of air under the same conditions is 76.38 pounds. It is thus evident that helium will have a lifting power of 65.82 pounds per 1,000 cubic feet in an air medium. The *Macon* held 6,500,000 cubic feet and had a gross lifting power of over 200 tons. Although slightly heavier than hydrogen, helium is preferable for use in lighter-than-air craft because it is noninflammable.

Helium closely approaches the theoretically perfect gas in its compressibility. It requires a temperature of 5.2° above absolute zero or minus 450.2° F. to liquefy it, and solidification can only be accomplished with extreme reduction of temperature and the application of high pressure.

The solubility of helium in water under standard conditions as reported by Hawkins and Shilling² is 0.0083 cc. per cc. of solution as compared to 0.0138 for nitrogen.³

USE IN MEDICINE

The pioneer work in this field was done by Barach⁴ who first tested the hypothesis of Hershey⁵ that exclusion of rare gases from the atmosphere was not compatible with life, and found it to be incorrect, for his animals lived "apparently uninfluenced in atmospheres in which the rare gases were excluded for periods as long as 42 days." In the same paper he reported on experiments confirming the work of Her-

² Hawkins, J. A., and Shilling, C. W. Helium solubility in blood at increased pressures. *J. Bio. Chem.*, 113: 649-653; 1936.

³ Hawkins, J. A., and Shilling, C. W. Nitrogen solubility in blood at increased air pressures. *J. Bio. Chem.*, 113: 273-278; 1936.

⁴ Barach, A. L. Rare gases not essential to life. *Science*, 80: 593; 1934.

⁵ Hershey, J. Willard. Physiological effects of oxygen atmospheres diluted by nitrogen. *Trans. Kansas Acad. of Science*, 32: 51-52; 1929.

they ^{6 7} and Sayers, Yant and Hildebrand ⁸ that helium was not itself harmful even when breathed in a mixture of 79 percent helium and 21 percent oxygen for 2 months. Helium was then proposed by Barach ⁹ as a therapeutic gas, for such a light gas mixture he says ¹⁰ "could be moved to and from the lungs more easily than air in those clinical conditions in which difficulty in air movement was present."

Barach ^{11 12 13 14 15} continued the work and suggested the use of helium-oxygen mixtures in the treatment of such conditions as asthma, obstructive lesion of the trachea, larynx and bronchi, emphysema, bronchiectasis, and pulmonary fibrosis. He pointed out ¹⁶ that—

In an attack of asthma the diameter of the small bronchi is reduced at first as a result of spasm of the constrictor muscles. The swiftly elevated negative pressure within the chest, which has become necessary to move air inward against an obstruction exerts a cupping action on the mucous membrane resulting in congestion and edema of the bronchial wall which further narrows the lumen of the smaller respiratory passageway.

The rationale for the use of helium-oxygen mixtures in the treatment of this condition rests in the decreased specific gravity of helium and in the fact that the pressure, and therefore the effort required for the movement of an 80-percent helium-20-percent oxygen mixture, is about one-half that required for air. The helium acts as the diluent and vehicle for the oxygen and goes through the constricted bronchi to the aveolar sacs where it is sorely needed to break the vicious cycle associated with the asthmatic attack. Also: ¹⁶

The sensation of dyspnea in patients with asthma may be mainly attributed to an increased effort on the part of the respiratory musculature to ventilate the lungs in the presence of narrowing of the smaller bronchi. The inhalation of a helium-oxygen mixture makes possible a more normal velocity of gas movement with diminished respiratory effort.

The use of helium-oxygen mixtures in asthma has continued to meet with signal success and has been confirmed and reported by Boothby,

⁶ Hershey, J. W. Components of air in relation to animal life. *Science*, **71**: 394-396; April 1930.

⁷ Hershey, J. Willard. Effects of synthetic atmospheres upon animal life. *Trans. Kansas Acad. of Science*, **34**: 240-243; 1931.

⁸ Sayers, R. R., Yant, W. & Hildebrand, J. Possibilities in the use of helium-oxygen mixtures as a mitigation of caisson disease. Report of Investigations, Dept. of Int. R. I. No. 2670; Feb. 1925.

⁹ Barach, A. L. Use of helium as a new therapeutic gas. *Proc. Soc. Exp. Bio. & Med.*, **32**: 462-464; 1934.

¹⁰ Barach, A. L. The effects of inhalation of helium mixed with oxygen on the mechanics of respiration. *J. Clin. Investigation*, **15**: 47-61; 1936.

¹¹ Barach, A. L. Helium used as a new therapeutic gas. *Anesth. and Analg.*, **14**: 210-215; 1935.

¹² Barach, A. L. Breathing of helium-oxygen mixture brings relief to asthma sufferers. *Science News-Letter*; 18 May 1935.

¹³ Barach, A. L. Use of helium in the treatment of asthma and obstructive lesions in the larynx and trachea. *Ann. Int. Med.*, **9**: 739-765; 1935.

¹⁴ Barach, Alvan L. Therapeutic use of helium. *J. A. M. A.*, **107**: 1273-1280; 1936.

¹⁵ Barach, A. L. Helium gas therapy. *Am. J. Surg.*, **34**: 588-590; 1936.

¹⁶ Barach, Alvan L. Physiological methods in the diagnosis and treatment of asthma and emphysema. *Ann. Int. Med.*, **12**: 454-481; 1938.

Mayo, and Lovelace;¹⁷ Goldsmith;¹⁸ Maytum;¹⁹ Maytum, Prickman, and Boothby;²⁰ Eyermann,²¹ and Metz, Wearner and Evans,²² who also recommend its use in pneumonia, unresolved pneumonia and cardiac decompensation. Kernan and Barach²³ report its use in cases of obstructive lesions in the trachea and larynx.

During 1938 and 1939 its use as an adjunct in the giving of general anesthesia was reported from the United States, England, and Mexico^{24 25 26 27}. In anesthesia it is considered of value in respiratory obstruction to economize effort and make respiration more effective, to prevent atelectasis, and as an aid in partial respiratory paralysis. At the Lahey Clinic, Eversole²⁸ reports:

It has also been helpful on many occasions by permitting us to continue smoothly and safely the administration of an anesthetic without inserting an intratracheal tube when otherwise one would have been necessary.

Helium and oxygen are used by Clough,²⁹ from the beginning, in the giving of cyclopropane general anesthesia; and Bonham³⁰ in discussing anesthetic allergy says that helium is a valuable adjunct and diluent for cyclopropane in the allergic cases, claiming that all true allergic cases were benefited from administration of helium. Behnke³¹ presents the physiologic considerations of inhalation anesthesia and helium.

The use of helium in artificial pneumothorax for pulmonary tuberculosis was reported abroad in 1930 by Reinders³² and again in 1938 by Schedtler.³³ Helium and oxygen mixtures have even been successfully added to an infant incubator by Benedict, White and Lee;³⁴ and

¹⁷ Boothby, Mayo and Lovelace. Oxygen and oxygen and helium therapy: recent advances. *Med. Clinics of N. A.*, **23**: 977-1005; 1939.

¹⁸ Goldsmith, N. Now, helium saves lives. *Scient. Am.*, **100**: 153; 1939.

¹⁹ Maytum, C. K. Helium and oxygen treatment of intractable asthma. *Proc. Staff Meet. Mayo Clin.*, **13**: 788-789; 1938.

²⁰ Maytum, Prickman & Boothby. Helium and oxygen in severe intractable asthma. *Proc. Staff Meet. Mayo Clin.*, **10**: 788-790; 1935.

²¹ Eyermann, Charles H. Diagnosis and treatment of bronchial asthma. *Diseases of the Chest*, **6**: 234-239; 1940.

²² Metz, C. W., Wearner, A. A., and Evans, A. E. Helium therapy (In status asthmaticus). *South. M. J.*, **32**: 34-40; 1939.

²³ Kernan, J. D., and Barach, A. L. Role of helium in cases of obstructive lesions in the trachea and larynx. *Arch. Otolaryng.*, **70**: 419-447; 1937.

²⁴ Bonham, R. F. Value as adjunct in general anesthesia. *Anesth. and Analg.*, **18**: 54-57; 1939.

²⁵ Sykes, W. S., and Lawrence, R. C. Helium in anesthesia. *Brit. M. J.*, **3**: 448-449; 1938.

²⁶ Vollbrechthausen, F. Helium: first use in Mexico as adjunct of general anesthesia. *Rev. Mex. de cir., ginec. y cancer*, **7**: 9-16; 1939.

²⁷ Vollbrechthausen, F. Helium in anesthesia. *Arch. med. ferroc.*, **1**: 159-165; 1939.

²⁸ Eversole, Urban. The use of helium in anesthesia. *J. A. M. A.*, **110**: 878-890; 1938.

²⁹ Clough, G. M. The use of helium. *M. J. Australia*, **2**: 400-402; 1939.

³⁰ Bonham, R. F. Cyclopropane anesthesia from allergic standpoint. (Dilution with helium.) *Anesth. and Analg.*, **18**: 288-291; 1939.

³¹ Behnke, A. R. Physiologic considerations of inhalation anesthesia and helium. *Anesth. & Analg.*, **19**: 35-41; 1940.

³² Reinders. Helium in artificial pneumothorax for pulmonary tuberculosis. *Nederl. tijdschr. v. geneesk.*, **74**: 6112; 1930.

³³ Schedtler. Use in pneumothorax. *Klin. Wchnschr.*, **17**: 1153-1154; 1938.

³⁴ Benedict, F. G., White, P., & Lee, R. C. An infant incubator employing controlled mixtures of helium and oxygen to combat respiratory failure. *Am. J. Obst. & Gynec.*, **39**: 63-70; 1940.

have been used by Cohn and Katzenelbogen in electro-encephalographic studies.³⁵

A simple method for the analysis of helium was reported by Schwenkner and Fallin;³⁶ and methods, apparatus and equipment for its use in inhalation therapy were reported by Eckman and Barach,³⁷ Lovelace,^{38 39} and by Matzger.⁴⁰

HELIUM-OXYGEN MIXTURES IN DIVING

It is the prevailing opinion of many that the use of helium in diving originated during the last 2 or 3 years, but as early as 1919 Elihu Thompson⁴¹ wrote to the Bureau of Mines suggesting its use in diving:

It is thought that by substituting helium for nitrogen in the air breathed, this depth (150 feet) might be say increased 50 percent or more.

His words were prophetic but he greatly underestimated the depth attainable. Four years later a patent was registered in the United States Patent Office by Charles Cooke⁴² for the use of helium with oxygen for divers, based on the fact that helium has a coefficient of solubility approximately half that of nitrogen, and because it is twice as diffusible.

Sayers, Yant, and Hildebrand⁸ in 1925 reported their pioneer work in the use of helium-oxygen mixtures as a mitigation of caisson disease. They said:

The substitution of helium for the nitrogen ordinarily present in the air we breathe has been found to result in an atmosphere which is as respirable as that provided by nature. The results obtained indicate that helium not only has the advantage of being less soluble than nitrogen, but also of diffusing more rapidly in the body fluids and tissues, which results in rapid elimination of the gas from the tissues during decompression. Along with mitigating the hazard of caisson illness, helium should markedly increase the scope of other kinds of engineering work in compressed air.

Concerning lessening the time necessary for decompression after breathing helium and oxygen under pressure they say:⁸

In a series of experiments on animals, after similar exposures decompression could be made from the helium-oxygen mixtures in as low as one-sixth the time

³⁵ Cohn, R., & Katzenelbogen, S. Electro-encephalographic studies. U. S. Nav. Med. Bull., 37: 596-599; 1939.

³⁶ Schwenkner, F. F., and Fallin, H. K. Helium. Simple method for analysis. Bull. Johns Hopkins Hosp., 61: 210-215; 1937.

³⁷ Eckman, Morris, and Barach, Alvan L. Inhalational therapy equipment. Modern Hospital, 57: 78-84; 1939.

³⁸ Lovelace, W. R., Jr. Oxygen for therapy and aviation: An apparatus for the administration of oxygen and helium by inhalation. Proc. Staff Meet. Mayo Clinic, 13: 646-654; 1938.

³⁹ Lovelace. Technic of treatment with helium and oxygen using B. L. B. inhalation apparatus. Proc. Staff Meet. Mayo Clinic, 13: 790-791; 1938.

⁴⁰ Matzger, E. Helium and oxygen mixture; economical and simple method for its use. Calif. & West. Med., 50: 418-419; 1939.

⁴¹ Thompson, Elihu. Correspondence. Science, 65: 36-38; 1927.

⁴² Cooke, Charles. U. S. Patent Office, 1923. Pat. No. 1,473,337.

necessary for air or nitrogen-oxygen mixtures. In a few preliminary experiments on men, decompression was made in one-fourth to one-eighth the time ordinarily recommended for air.

The advantage which helium has been found to possess may be employed either through supplying the men with the synthetic air throughout the diving period or during a shorter period at the end including the decompression. It also might be used as a wash gas during decompression only or as a treatment if compressed air illness has occurred with ordinary air, the benefits being due to the removal of the partial pressure of nitrogen in the medium that is removing the gas.

In 1926 Sayers and Yant ⁴³ and in 1927 Yant ⁴⁴ reported on the use of helium-oxygen atmospheres in deep-sea diving and caisson operations. In the latter year this Bulletin ⁴⁵ reported briefly on helium-oxygen mixtures in diving.

Stewart ¹ in his excellent paper on helium says:

Divers and caisson workers can work for more extended periods and under higher pressure with greatly increased safety and comfort, and may be returned to normal pressure conditions in a fraction of the time required when compressed air is the atmosphere breathed while at work.

He reasoned that this is because helium is extremely insoluble. It was to determine definitely the relative solubility at atmospheric and increased pressures that Hawkins and Shilling studied the solubility of nitrogen ³ and of helium ² in blood and found that the amount of either nitrogen or helium dissolved by whole blood under pressures varying from 1 to 6 atmospheres (absolute) was directly proportional to the pressure of the gas according to Henry's law, and that the solubility of helium under standard conditions was 0.0083 cc. per cc. of solution, while that of nitrogen was 0.0138. The English physiologist G. W. M. Boycott wrote to Capt. John D. Craig in January 1940 and enclosed a proposal which he made in 1935 concerning the breathing of helium-oxygen during the period of decompression following a dive. He states ⁴⁶: "The rates of evolution of a gas from a liquid are the same whether the evolution takes place into a vacuum or into a closed vessel containing another gas" and from this he reasons that the breathing of helium following a dive would "double the rate at which the nitrogen comes out of solution."

Up to this time, those reporting the use of helium-oxygen had stressed its value in allowing the decompression time to be materially shortened while at the same time the danger of compressed-air illness was lessened; but it remained for Dr. Edgar End ⁴⁷ in 1937 to point out probably the most important use of helium in diving—which is in the

⁴³ Sayers, R. R., & Yant, W. P. Value of helium-oxygen atmosphere in diving and caisson operations. *Anesth. & Analg.*, 5: 127-138; 1926.

⁴⁴ Yant, W. P. Helium in deep-sea diving. *Ind. & Eng. Chem., News Ed.*, 5: 4; 1927.

⁴⁵ Notes & Comments. Helium-oxygen mixture in diving. *U. S. Nav. Med. Bull.*, 25: 154; 1927.

⁴⁶ Boycott, G. W. M. Principle of the partial pressure of gases in its application to the Haldane method of stage decompression. Unpublished paper sent to Captain Craig. 1940.

⁴⁷ End, Edgar. Rapid decompression following inhalation of helium-oxygen mixture under pressure. *Am. J. Physiol.*, 120: 712-718; 1937.

lessening, and in some cases elimination, of the untoward mental and neuromuscular symptoms observed under increased air pressure. These symptoms, reported by Behnke, Thomson, and Motley ⁴⁸ and studied and reported by Shilling and Willgrube,⁴⁹ range all the way from slight dizziness to complete loss of consciousness and are experienced by all men working in high-pressure air. This narcotic effect of greatly increased air pressure has been the limiting factor in the depth which divers could attain. End ⁴⁷ said:

It is my belief that in addition to shortening the time necessary for safe decompression, helium may also free divers from the untoward psychological effects of air at high pressure.

The following year, 1938, in reporting a world-record dive of 420 feet in a flexible diving suit breathing helium-oxygen, End ⁵⁰ says:

At no time were there noted any mental changes such as have been described in men breathing compressed air under great pressure, and it is speculated that this freedom from mental change may have been due to the nature of the helium-oxygen mixture employed.

In the same year Behnke and Yarbrough ⁵¹ reported on physiologic studies of helium and said:

Essentially the helium-oxygen atmosphere abolishes, or renders negligible, the stupefaction and impaired motor control associated with air respiration under pressure.

Again ⁵² they studied the mental effects of argon compared with helium and nitrogen, and found them to be greatest under argon and least under helium. At the same time Momsen ⁵³ reported on the use of helium-oxygen mixtures for diving. Both of these reports were on work done at the Experimental Diving Unit in Washington, D. C., prior to the sinking of the U. S. S. *Squalus*. At about the same time, Ellsberg ⁵⁴ wrote a popular story of the helium work being done in the United States Navy. Recently End ⁵⁵ reviewed the physiologic effects of increased pressure and stated:

The most important development arising from a desire to shorten decompression time has been the introduction of artificial breathing mixtures for men under pressure. The most successful of these has been a mixture of helium and oxygen.

⁴⁸ Behnke, A. R., Thomson, R. M., and Motley, E. P. Psychologic effects from breathing air at 4 atmospheres pressure. *Am. J. Physiol.*, **112**: 554-558; 1935.

⁴⁹ Shilling, C. W., and Willgrube, W. W. Quantitative study of mental and neuromuscular reactions as influenced by increased air pressure. *U. S. Nav. Med. Bull.*, **35**: 373-380; 1937.

⁵⁰ End, Edgar. Use of new equipment and helium gas in a world-record dive. *J. Industrial Hyg. and Tox.* **20**: 511-520; 1938.

⁵¹ Behnke, A. R., and Yarbrough, O. D. Physiologic studies of helium. *U. S. Nav. Med. Bull.*, **30**: 542-558; 1938.

⁵² Behnke and Yarbrough. Respiratory resistance, oil-water solubility and mental effects of argon compared with helium and nitrogen. *Am. J. Physiol.* **126**: 409-415; 1939.

⁵³ Momsen, C. B. General discussion—Use of helium-oxygen mixtures for diving. Report on use of helium mixtures for diving, E. D. U., Navy Yard, Washington, D. C.; April 1939.

⁵⁴ Ellsberg, Ed. Diving gas. *Collier's*, pp. 22-28, April 15, 1939.

⁵⁵ End, Edgar. The physiologic effects of increased pressure. Submitted for publication.

Anyone working with helium at once notices the peculiar voice change produced by breathing the gas. This has been explained as due to the fact that we unconsciously set the muscles of our larynx to cause a certain sound in air and when breathing a medium so much lighter, the sound produced is much changed from normal.⁵⁶

Another factor of interest, and of much greater importance, was the report by Lovelace, Mayo and Boothby⁵⁷ of the use of helium-oxygen inhalations to prevent "aero-otitis media" caused by the lack of ventilation of the middle ear during changes in atmospheric pressure to such an extent that traumatization occurs in the tympanic cavity. As in exposure to increased air pressure in diving, it is due to stenosis of the eustachian tube caused by disease or inflammation. The divers say they are "blocked," "can't clear their ears," or "can't equalize pressure." The authors found that in most aviators or airplane passengers the condition could be prevented or alleviated by breathing a mixture of helium-oxygen. Again this is due to the lightness of the gas and its rapid rate of diffusion (2.7 times that of nitrogen) and to the greatly increased mean velocity of the helium molecule. A similar report was made by Henrich⁵⁸ and by Lovelace.³⁸ Of course the same beneficial results are obtained when, instead of coming from rarified air to atmospheric pressure, one is going from atmospheric pressure to increased air pressure. Levy (personal conversation with the writer) told of its successful use in the New York City tunnel operations in clearing "blocked" ears.

The salvage of the U. S. S. *Squalus* gave ample proof of the value of the use of helium in deep-sea diving. It is the opinion of all that this monumental piece of salvage work could not have been accomplished without the use of helium. Behnke and Willmon⁵⁹ say:

In contrast with previous diving methods, the distinguishing features of the diving technic were the successful employment of helium-oxygen mixtures for deep diving in cold water, made necessary by the failure of the standard method of using air * * *.

Much work remains to be done relative to the use of helium-oxygen mixtures in the treatment of medical conditions, as well as in the field of deep-sea diving and work under greatly increased air pressure, but the ground work has been well laid for those who are now interested in extending its use.

⁵⁶ Editor. Voice changes from inhalation of He-O₂. J. A. M. A., 113: 706; 1939.

⁵⁷ Lovelace, Mayo and Boothby. Aero-Otitis Media: its alleviation or prevention by the inhalation of helium and oxygen. Proc. Staff Meetings Mayo Clinic, 14: 91-96; 1939.

⁵⁸ Henrich, F. Helium Application in aviation and medicine. Sitzungsber. d.phys-med. Soz. zu Erlangen, 70: 385-386; 1939.

⁵⁹ Behnke, A. R., and Willmon, T. L. U. S. S. *Squalus*. Medical aspects of the rescue and salvage operations and the use of oxygen in deep-sea diving. U. S. Nav. Med. Bull., 37: 629-640; 1939.

ROUTINE KAHN BLOOD REACTIONS

REPORT OF 10,000 TESTS MADE ON NAVAL RECRUITS¹

By Captain G. E. Thomas, Medical Corps, United States Navy, and Lieutenant (Jr. Gr.) E. W. Garrity, Medical Corps, United States Navy

This report is based on ten thousand routine Kahn tests on recruits entering the Naval Training Station, San Diego, California, between July 1939 and July 1940. These recruits are drawn from all the states west of the Mississippi River except Minnesota, Missouri, Kansas, Nebraska, Iowa, and the Dakotas, also from Mississippi and Louisiana.

The tests in this group have been read as positive, doubtful or negative in conformity with the instructions of the majority of authorities at this time. Our tests are all run in the approved method and results have been checked against the laboratories of the United States Naval Medical School, the San Diego Naval Hospital and the University of Michigan Hospital.

In ten thousand tests there were found 12 persistently positive blood Kahns. Ten of these men have been discharged from the naval service and two are awaiting disposition. This is an incidence of .12 percent. There were no other physical findings in these men that would indicate that their persistent positives were due to any disease other than syphilis.

Aside from the persistently strong positives, there were 26 strongly positive Kahns which were characterized as false positives by subsequent negative findings. All false positive Kahns returned to negative within 3 to 4 weeks after the original positive.

In addition to the above, there were found 16 weakly positive or doubtful Kahns which became negative within a day or so after the weak positive. These doubtful tests which were not regarded as false positives leave the incidence of false positives at 0.26 percent.

INFLUENCE OF VACCINATION WITH COWPOX

In this series of 26 false positives, it is believed that there is a possible relationship to reactions following cowpox vaccination. Among the entire group of men receiving cowpox there were 2,614 primary reactions, 2,589 accelerated reactions and 4,797 immune reactions. The accelerated reaction is a modified primary reaction and represents all degrees from immunity to vaccinia. Considering the accelerated and primary reactions together, we found that there was an incidence for the entire group of 52 percent. Among the 26 false positive cases, there were 11 primary reactions, 12 accelerated reactions and only 3 immune reactions, which give an incidence of 88 percent for the 23 false positive cases showing primary or accelerated reactions.

Considering only the primary reactions, we find that among the men having false positive Kahns there were 42 percent primary

¹ From the U. S. Naval Training Station, San Diego, Calif.

reactions while in the entire group of 10,000 men there were only 26 percent primary reactions.

From the above results it is believed that primary and accelerated cowpox reactions may have some effect on the Kahn test.

Barnard of Chicago² mentions a case which had strongly positive Kahns for 4 weeks following vaccinia. This patient had no evidence of syphilis and his blood Kahn subsequently became negative. Barnard seems to believe that the vaccination result definitely affected the Kahn test.

INFLUENCE OF ANTITYPHOID INOCULATION

There apparently is no relationship between antityphoid inoculation and the positive reactions of the Kahn test. Exclusive of the persistent Kahns, there were 9 false-positives after cowpox and before the first inoculation, 2 after cowpox and one inoculation, 2 after cowpox and two inoculations and 13 after cowpox and 3 inoculations. There were no serious reactions in this group which had false-positive Kahns and only 2 or 3 mild reactions. The inoculation reaction generally lasts only 24 hours and with the exception of the man noted below, all bloods for Kahn tests were drawn 48 hours or more after antityphoid inoculations. The 1 man found in the positive Kahn group, who had blood drawn on the day following his antityphoid inoculation, had only a mild reaction and proved to be a persistent positive case.

Kolmer has cited the following conditions as having a possible effect on the diagnostic test: Leprosy, malaria, relapsing fever, pellagra, scarlatina, and yaws depending on how one views the relationship between the last-named disease and syphilis. Stokes has added acute bacterial endocarditis, acute infectious mononucleosis and certain other febrile diseases. Others have reported Vincent's, pneumonia, tuberculosis, chancroid, lymphopathia venereum, trypanosomiasis, rheumatic fever, typhus fever, Leishmaniasis, malignancy (especially carcinoma of the tongue), glanders, Weils disease, and injections of horse serum as having some effect on the diagnostic test. In addition there is some belief that there is a reagin-like factor in normal human blood.

In connection with the malarial aspect, it may be added that one recruit had a negative Kahn upon arrival, subsequently developed an acute relapse of a previous malarial infection and had a weakly positive blood Kahn. After treatment started the Kahn test became negative.

Mitchell of Denver³ reviewed the value of the diagnostic tests and stated that the Kahn test was of exceptional value because of the

² Barnard, R. D.: False positive blood serological tests for syphilis following vaccination for variola. *Ill. Med. Journ.*, 77: 78-79, January 1940.

³ Mitchell, W. C.: Serodiagnostics of syphilis with particular reference to Kahn test. *Colo. Med.*, 24: 311-316, May 1937.

scarcity of false positive results which it gave. He mentioned the results during the League of Nations trials of 1928 in Copenhagen in which 957 Kahns gave no false positives and noted that similar results were found at Montevideo in 1930 and by the United States Public Health Service in 1935.

Among the persistent positive cases of which 10 were discharged from the service and 2 await disposition, all of which had their blood checked by another laboratory and by two different diagnostic tests, the following decisions are now reached: 4 were believed to have had acquired syphilis, 2 were believed to have had congenital syphilis, 4 were persistently seropositive and had histories and physical findings which were suggestive, but not definite, of acquired syphilis, and 2 were definitely doubtful.

Beginning in July 1940, bloods for Kahn tests will be drawn prior to cowpox vaccination and typhoid inoculation. These results measured against the above findings will clarify our now insecure conclusions.

CONCLUSIONS

1. On the basis of our findings it is believed that primary and accelerated cowpox reactions have some effect on the diagnostic Kahn test.

2. We have been unable to determine any relationship between antityphoid inoculation and the diagnostic Kahn test.

GONORRHEA, 1941

By Commander E. P. Parsons, Medical Corps, United States Navy

It is now possible to say in remarkably precise terms how much has been gained by the introduction of drugs of the sulfanilamide group in the treatment of gonorrhea. The problem of gonorrhea and its treatment is as old as the human race, but until 1937 the solution remained about as vague as anything in clinical medicine. True, there were drugs and measures of unquestioned value but these values were not definite enough to express in numerical terms. Before 1937 the methods and measures were so poorly standardized and their results so generally disappointing that only the roughest sorts of estimates could be given to their values.

It takes a long time to evaluate a drug. We must have an exact cross section made up of the clinical courses of thousands of cases over a long trial period for the drug and another cross section for a control, similarly constructed for a similar period without the drug. When sulfanilamide first appeared many wild statements and guesses, some wildly optimistic and others dismally pessimistic, were made concerning its worth.

Unless it is in the Army figures, there are quite certainly no statistical tables pertaining to gonorrhea more reliable than those to be found in the annual reports of the Surgeon General, United States Navy. These tables represent large case numbers, and because of regulations covering the reporting of all complications, sick days, etc., they reach a high accuracy of detail for a disease in which many peculiarities, mostly of the social sphere, prevent any but the loosest sort of statistical collections or reports in the civilian world.

What imperfections exist in the Navy statistics are in the absolute levels of incidence and not in their relative heights from year to year. Thus, because of concealments, failures of diagnosis and other factors, the levels reported for original admissions and for the various complications are always slightly less than the truth, but as these factors continue without appreciable change from year to year, the range of annual fluctuations for such items as the frequency of the various complications, their respective average sick days per case, percent of invaliding from the service, etc., are almost exactly as shown in the reports. One of the intriguing features of gonorrhea is the almost mathematical precision of the ratio in which certain complications will arise in any large number of cases, the constancy of the average sick days per case for any large number of uncomplicated cases, or for any given complication, and the invariability of the percentage of cases that will remain chronic or that will tend to recur or that will result in invaliding from the service. To illustrate: (a) The percentage of new infections which became complicated by epididymitis in 1934, 1935, and 1936 were 14.2, 15.5, and 15.9 respectively, (b) the percentages for all forms of complications for those years were 20.3, 22.6, and 22.6 respectively, (c) sick days per case of epididymitis for those years were 30.0, 31.1, and 28.9 respectively and for all other complications 49.0, 48.2, and 47.4 respectively, (d) sick days per case of original admissions, all forms, simple and complicated were 16.5, 17.1, and 19.0 respectively, and (e) service invalidings percentage were 1.3, 1.0, and 0.8 respectively. Note that in each series there is scarcely a fluctuation of one point up or down from the middle value of the three. If the levels for these items were to be charted graphically over any group of years prior to 1937 they would have to be represented by essentially straight horizontal lines.

All these things being true, then if some new drug or method of remarkable repute is introduced, used extensively and with reasonable intelligence, the previously horizontal lines should take a dip corresponding in degree to the merit of the new therapeutic measures. Very conveniently and happily for the purposes of this study, sulfanilamide came into wide use about the middle of the year 1937. The year 1937 should then give us intermediate values, since the drug

was used for just half a year, as compared to the 1938-39 2-year period when it was used extensively throughout the entire period and as compared to the immediately preceding 2-year period of 1935-36 when it was not used at all. Reference to the following tables reveals that this is exactly what happened.

TABLE 1.—*Complication percentages*

Year	Original admissions		Complications of original admissions						Invalidated from service	
			Epididymitis		All others		Total			
	Number	Rate per 1,000	Number	Percent	Number	Percent	Number	Percent	Number	Percent
1935.....	5,811	51.4	903	15.5	411	7.0	1,314	22.6	63	1.0
1936.....	5,400	43.8	862	15.9	365	6.7	1,227	22.6	48	.8
1937.....	5,778	43.7	717	12.4	394	6.8	1,111	19.2	40	.6
1938.....	7,260	58.9	595	8.2	307	4.2	902	12.5	30	.4
1939.....	8,554	57.1	706	8.2	332	3.8	1,038	12.2	20	.2

TABLE 2.—*Sick days*

Year	Urethritis, un- complicated		Urethritis with complications						Total	
			Epididymitis		All others		Total			
	Number	Average per case	Number	Average per case	Number	Average per case	Number	Average per case	Number	Average per case
1935.....	51, 827	8. 9	28, 105	31. 1	19, 829	48. 2	47, 934	36. 4	99, 761	17. 1
1936.....	60, 951	11. 2	24, 615	28. 9	17, 317	47. 4	41, 932	34. 1	102, 883	19. 0
1937.....	54, 813	9. 5	20, 194	28. 1	18, 733	47. 5	38, 927	35. 3	93, 740	16. 2
1938.....	60, 401	8. 3	15, 738	23. 0	12, 549	40. 8	28, 287	31. 3	88, 688	12. 2
1939.....	74, 205	8. 6	18, 648	26. 4	13, 893	41. 8	32, 541	31. 3	106, 746	12. 3

TABLE 3.—*Reduction in complications noted in recent periods*

Period	Original admissions for urethritis	Urethritis with complications					
		Epididymitis		All others		Total	
		Number	Percent	Number	Percent	Number	Percent
1935-36.....	11,211	1,765	15.7	776	6.9	2,541	22.6
1937.....	5,778	717	12.4	394	6.8	1,111	19.2
1938-39.....	15,814	1,301	8.2	649	4.4	1,940	12.6
Percent of reduction.....			47.7		36.2		44.2

TABLE 4.—*Reduction in sick days noted in recent periods*

Period	Urethritis, uncomplicated		Urethritis with complications						Combined total		Invalidated from service	
			Epididymi- tis		All others		Total cases					
	Total sick days	Aver- age per case	Total sick days	Aver- age per case	Total sick days	Aver- age per case	Total sick days	Aver- age per case	Total sick days	Aver- age per case	Cas- es	Per- cent
1935-36	112,778	10.0	52,717	29.8	37,146	47.8	89,863	35.3	202,644	18.0	111	0.9
1937	54,813	9.5	20,194	28.1	18,733	47.5	38,927	35.3	93,740	16.2	40	.6
1938-39	134,606	8.5	34,386	26.4	26,442	41.4	60,828	31.4	194,434	12.3	50	.3
Percent of reduction		15.0		11.4		13.3		11.0		31.6		66.6

Sulfapyridine and sulfathiazole did not come into use until 1940, so that the gains accomplished from the pre-1937 to the post-1937 2-year periods should be credited entirely to sulfanilamide. Reference to table 1 will show that the curves remained on their new horizontal levels during the 1938-39 period, that is, the 1939 figures remained the same as the 1938 figures. It will, of course, be of particular interest to see what further dips they will take and what new horizontals they will run on during the 1940-41 period under the influence of sulfapyridine and sulfathiazole and possibly something even better than these two drugs and not heard of at this writing. The figures for these new and unquestionably better drugs will not be available until 1942. We should expect 1941 to show much better figures than 1940, since sulfapyridine did not come into vogue until almost the middle of 1940. The year 1940 should show intermediate figures between those of 1939, when no sulfapyridine was used, and those of 1941 when sulfanilamide will be, we hope, entirely replaced by the far superior sulfapyridine, and perhaps by sulfapyridine's apparent peer, sulfathiazole.

Let us sum up here the analysis of the progress made by sulfanilamide:

(1) The epididymitis complication percentage dipped from 15.7 to the intermediate (1937) level of 12.4 and then on to the new level of 8.2, a reduction of 47.7 percent in the frequency of epididymitis occurrences.

(2) The other complications as a group (joint, prostate, etc.) underwent a similar drop with corresponding figures of 6.9, 6.8, and 4.4, a reduction of 36.2 percent.

(3) The corresponding figures for total complications are 22.6, 19.2, and 12.6, a reduction of 44.2 percent.

(4) Sick days per case were reduced in each division of gonococcal infections, as follows:

	Percent reduction
Urethra 10.0, 9.5, and 8.5	15.0
Epididymis 29.8, 28.1, and 26.4	11.4
Other complications 47.8, 47.5, and 41.4	13.3
Total complications 35.3, 35.3, and 31.4	11.0
Total, all forms 18.0, 16.2, and 12.3	31.6

(5) The largest reduction is to be found in the percentage of new admissions which resulted in invalidings from the service. These were 0.9, 0.6, and 0.3. Reduction—66.6 percent.

(6) In general terms it can be said that the use of sulfanilamide as compared to methods of treatment prior to 1937 reduced complications by nearly one-half, sick days per case, all forms, by nearly one-third and service invalidings by nearly two-thirds.

The new drugs have wrought in the gonorrhea picture other changes which can be described in terms less mathematical than those used above and in terms more readable and more pleasant to contemplate.

Gonorrhea, which formerly constituted a rather formidable problem and pretty much of a headache for the Medical Department has now reached the status of a minor nuisance. The old familiar scenes of crowds of men lining up at the trough with their argyrol syringes and permanganate irrigating jars have vanished and we do not regret their departure. Looking back on those scenes, so unlike the "Old Oaken Bucket," brings forth anything but "fond recollections so dear to our hearts." We used to keep a 25-gallon collection of double strength stock solution of permanganate on a shelf in our treatment room at a naval hospital and this had to be replenished about twice a month. Now we use a 5-gallon bottle, refill it about once a month and use it only for irrigations in prostatic surgical cases. As for the argyrol syringes and the time-honored black watering trough, they have long since been completely abandoned.

No longer do we wonder whether the patient will ever recover from his chronic gonorrhea, or worry about how long it will be before he has his next recurrence; no longer are we forced to discharge him from the navy because it appears that he will never get well from his gonorrhea. No longer is the urological department of a naval hospital principally a venereal ward or referred to as "the clap ward." Since 1937 the urological wards of the present writer have, despite a constant increase in the general volume of work, maintained the proportion of gonorrhea patients always in a minority status of the general ward population. We may soon reach the point where naval hospitals may go for days at a time without harboring a single gonorrhea patient. This condition actually occurred for a period of 2 weeks in July 1938 at the Pearl Harbor naval hospital. It almost occurred recently at the Washington naval hospital. The last remaining gonorrhea patient was leaving to return to duty on a certain day, but 3 hours before he left another gonorrhea patient was admitted. The Pearl Harbor incident is probably the first of its kind in the history of naval hospitals.

If we could all take a saner view of gonorrhea we could reduce the sick days and other inconveniences of the disease far beyond what has already been accomplished by the new drugs. It has always seemed to this writer a senseless procedure to relieve an uncomplicated case of gonorrhea from regular duty and an unreasonable procedure to

transfer him to the hospital. This amounts to kicking a man when he is down. He is surely down in spirits when he discovers his infection and to add to this injury the insult of taking away his pay when he is able and willing to work is a very low act. Every time a case of uncomplicated gonorrhea is transferred to a hospital it is a serious reflection on the medical officer who makes the transfer because it arouses the strong and usually warranted suspicion that he is unwilling to assume his responsibilities or his work. In spite of what has just been said, uncomplicated cases were kept on the sick list for a total of 74,205 days in 1939. This amounted to an average of 8.6 days for every new admission for gonorrhea during the year.

Frequently the argument of isolation is used as an excuse for admission to the sick list or transfer to a hospital. Transmission of gonococcal infection from male to male is practically unheard of and it is doubtful if it happens once in a million infections. The absurdity of isolation of males from males is therefore obvious. The removal of mess attendants, cooks, or bakers from their duties and their retention on the sick list because of gonorrhea is a procedure that can have no support in our knowledge of the mode of transmission of gonorrhea. If we do it on the ground of our esthetic sensibilities then we could profitably trade some sensibility for some sense.

Restriction, however, is quite a different matter. It accomplishes three important things: (a) It isolates the infection from the all important intermediate host, the opposite sex, thus preventing dissemination of the infection among the civilian population and ultimately prevents its return to the navy, (b) it prevents alcoholic indulgence, (c) and it prevents sex excitation. It even accomplishes a fourth purpose, if it pleases those who believe in punitive measures. Two months after apparent recovery should be the very minimum of the restriction period if it is to accomplish the first three purposes mentioned. Three months would be a safer period. This period should give the man plenty of time to contemplate the importance of proper prophylaxis; to contemplate the disasters that may strike those who fail to take the recommended precautions, either mechanical protection or immediate and thorough chemical prophylaxis.

This leads naturally to the long mooted subject of prophylaxis. The entire question seems to hinge on the simple fact that no method, even a 100 percent perfect method, is any good unless it is used in 100 percent of the user's exposures. More than that, it must be used properly and in the case of chemical measures it must be used immediately. When prophylaxis is used tardily, haphazardly or only in occasional instances of one's exposures it becomes nothing more than a sort of superfluous ritual, something like the use of the finger bowl at the conclusion of a meal; and that is what it appears to be, for the most part, in the U. S. Navy at present. The question arises as to

whether it might not be better if we abolished prophylaxis facilities on ships and shore stations and furnish the men only with the facilities which could be used, as they should be, at the scene and time of the exposure. This might impress them with the futility of late and the necessity of early prophylaxis.

Unconfirmed reports are at hand of the efficacy of sulfanilamide as a prophylactic when used in the following way: 30 grains in one dose 3 to 4 hours before exposure and another 30-grain dose the next morning.

The responsibility for a good prophylaxis program on any ship or station rests only partially with the men. Good instruction, especially at recruit training stations, together with other antivenereal measures is largely a medical department responsibility and presupposes strong support and cooperation on the part of the line authorities. Any relaxation or other weak link in the chain of the program soon results in an increased incidence of venereal infections and this in turn is a reflection on both the line and medical officers of the ship or station concerned. In Hugh Young's recent autobiography he tells of the early difficulties of his antivenereal efforts with the A. E. F. in France. After General Pershing issued certain drastic orders pertaining to the campaign that Young had outlined, and which incidentally included the holding of line officers responsible for the venereal rates under their commands, the venereal rate of the A. E. F. fell from one of the highest in military history to the lowest that has ever been achieved.

A FORMULA FOR IMMEDIATE CHEMICAL PROPHYLAXIS

SUPPLEMENTARY REPORT

By Rear Admiral H. W. Smith, Medical Corps, United States Navy

In 1938 there was published in this BULLETIN a progress report of work being done in connection with the formulation of a unitary prophylactic ointment.¹

Since then, further work has suggested changes in certain of the ingredients—for the most part, substitutions made in the interest of economy or to facilitate large-scale production.

For example, d-sorbitol has been substituted for cholesterol because, while equally suitable and efficient, it costs very much less; and other emulsifying agents may be used in the future as required by current availability and prevailing prices: e. g., mannitol or mannitan mono-palmitate. The use of one emulsifier or another involves no alteration in the character or activity of the finished product.

Since it has been found that a few persons experience slight burning following injection into the urethra of ointment containing mercuric

¹ Smith, H. W., U. S. Nav. Med. Bull. 36: 522, October 1938.

cyanide, oxyquinoline benzoate has been selected as the nonspecific antiseptic, this in order to obviate a possible objection to use of the preparation on the score of irritation. Oxyquinoline, however, like cholesterol is expensive and some other antiseptic may be substituted if search discovers one cheap and equally suitable.

SULFANILAMIDE AND RELATED PRODUCTS

SUPPLEMENTAL REPORT ON CHEMOTHERAPY II

By Lieutenant Robert A. Bell, Medical Corps, United States Navy

The use of sulfanilamide and related products has become widespread since their recent introduction. The distribution of over 300 tons of the drugs in the past year gives an indication of this volume.

A detailed report was presented in the January 1939 issue of this publication by a number of naval medical officers. These few inter-

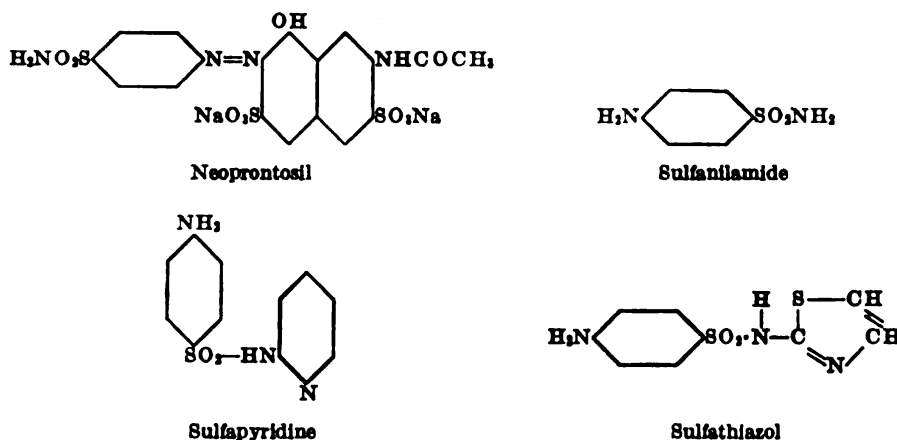


FIGURE 1.—Structural formulas of compounds indicated.

vening months have seen the introduction of many new compounds, of controversial ideas regarding the proper time for instituting this therapy, of some determination of the indications for the use of each drug, and of some degree of confidence in foreseeing toxic reactions. This wealth of accumulated experience has warranted this résumé which is intended only to supplement, from the general literature, the reports previously presented in this BULLETIN. For this reason the present discussion is incomplete unless reviewed with this relationship in mind.

Of all the compounds synthesized, only a few are widely used. The use of Neoprontosil is advocated especially because of its low toxicity which enables it to be used for long periods. It is rapidly absorbed from the intestinal tract, rarely induces serious toxic reactions, and is well adapted for treatment of such conditions as chronic ulcerative colitis requiring prolonged therapy.

Sulfanilamide has been widely used, is well known, and is relatively nontoxic. It is highly effective against streptococci of Lancefield's group A, less effective for group B and only moderately effective against group C. It has some effect against *salivarius* and *mitis* strains of the *alpha* hemolytic streptococci, excepting in subacute bacterial endocarditis. It is effective against the streptobacillus of Ducrey, the meningococcus, gonococcus, *C. welchii* and the usual urinary tract invaders, notably *B. coli* and *P. vulgaris*. It has some as yet undetermined degree of effectiveness against *Brucella* organisms and the viruses of lymphogranuloma venereum and trachoma. It is apparently valueless in enterococcic infections.

The pyridine derivative¹ (2-sulfanilyl aminopyridine, Dagenan, "M&B 693"), sulfapyridine is less regularly absorbed from the gastro-intestinal tract than is sulfanilamide. It resembles sulfanilamide in its ready penetration of all tissues and body fluids in concentration not far removed from that of the blood. However, unlike sulfanilamide, it is usually present in higher concentration in the liver (in animal experiments) than in other tissues and there is more variability in its distribution from animal to animal. It induces severe nausea in many patients and because of its low solubility tends to crystallize in the renal tubules producing hematuria and anuria. It has proven strikingly effective in pneumococcic pneumonia and in other pneumococcus infections such as otitis, mastoiditis, and meningitis and is superior to sulfanilamide in gonococcus, staphylococcus, and Friedlander's bacillary infections.

The thiazole analogue of sulfapyridine, sulfathiazole, synthesized by Fosbinder and Walter² and Lott and Bergeim,³ is receiving intensive clinical trial. It has recently been released to the medical profession after being referred to the Pure Food and Drug Administration. It appears to be less toxic, more soluble, and more readily absorbed than sulfapyridine.⁴ Metabolic studies indicate it is more rapidly metabolized and undergoes much less conjugation than sulfapyridine. It is not reabsorbed from the renal tubules as readily as sulfanilamide⁵ or sulfapyridine but it is more soluble than sulfapyridine and renal concretions are less likely. It is the most effective

¹ The efficacy of this drug against the pneumococcus was first determined by Whitby (Chemotherapy of pneumococcal and other infections with 2-(p-aminobenzenesulfonamido) pyridine, *Lancet*, 1: 1210-1212, May 28, 1938), who referred to its laboratory serial number "M&B 693." It was first made available to physicians in England by May & Baker, Ltd., who gave it the brand name "Dagenan." It was introduced in the United States by Merck & Co., Inc., and is now known as sulfapyridine.

² Fosbinder, R. J., and Walter, L. A.: Sulfamido derivatives of heterocyclic amines, *Jr. Am. Chem. Soc.*, **61**: 2032, 1939.

³ Lott, W. A., and Bergeim, F. H.: 2(p-aminobenzenesulfonamido) thiazole: a new chemotherapeutic agent, *Jr. Am. Chem. Soc.*, **61**: 3593, 1939.

⁴ McKee, C. M., Rake, G., Greep, R. O. and Van Dyke, H. B.: Therapeutic effect of sulfathiazole and sulfapyridine, *Proc. Soc. Exper. Biol. and Med.*, **42**: 417, November 1939.

⁵ Reinhold, John G., Flippin, H. F. and Schwartz, L.: Observations on the pharmacology and toxicology of sulfathiazole in man, *Am. J. M. Sc.*, **199**: 393-401, March 1940.

drug in staphylococcus infections,^{4,8} is apparently superior to sulfapyridine in gonococcus infections, equal to it in experimental⁴ and clinical⁶ pneumococcus infections and is effective against *S. faecalis* organisms in the urinary tract.⁷

The drugs disulfanilamide and dimethylsulfanilamide were never marketed in this country because of the high percentage of peripheral neuritis which they induce. Similarly sulfamethylthiazole has been withdrawn from experimental clinical use for the same reason, though this drug is very effective against staphylococcus infections, and proved, at least in the hands of some workers, to cause peripheral neuritis only occasionally.

At the present time a combination of sulfanilamide and pyrimidine, a product of the Stamford Research Laboratories of the American Cyanamid Company, is receiving experimental and clinical evaluation. It is labeled sulfadiazine and appears to be more soluble, more readily absorbed, less toxic and less readily converted to the acetyl derivative than is sulfapyridine or sulfathiazole.

The method of action of these compounds against bacteria has been the subject of much work and debate. A direct bactericidal effect has not been satisfactorily demonstrated. It is apparent that some degree of direct bacteriostatic effect does occur. Whether this is the most important function is not known. Yet any bacteriostasis will inhibit, to some extent, the production of bacterial toxins and thereby limit bacterial toxicity, invasiveness, and tissue destruction. Under these conditions the immune mechanisms of the host are allowed to develop under more favorable conditions and to maintain a more effective check on the invader. This does not imply any direct inhibition of the formation of bacterial toxins nor has there been any anti-toxic effect noted.^{9 10 11 12 13}

The clinical course and mortality rates of many diseases are markedly reduced by this therapy. This course of events is further altered favorably by the administration of immune sera and antitoxins. If insufficient immune sera and antitoxin are given, and the drug discontinued before host immunity has developed, the resultant relapse is familiar in cases infected with the pneumococcus, streptococcus or meningococcus. Thus host immunity proves the decisive factor. The process is typified in pneumococcus pneumonia cases

⁴ Flippin, H. F., Schwartz, L. and Rose, S. B.: Comparative effectiveness and toxicity of sulfathiazole and sulfapyridine in pneumococcal pneumonia, *Annals of Int. Med.*, **13**: 2038-2050, May 1940.

⁷ Pool, F. L. and Cook, E. N.: Sulfathiazol and sulfamethylthiazol in the treatment of infections of the urinary tract, *Proc. Staff Meet. Mayo Clinic*, **15**: 113-117, February 21, 1940.

⁸ Barlow, O. W., and Homburger, E., Specific chemotherapy of experimental staphylococcus infections with thiazol derivatives of sulfanilamide, *Proc. Soc. Exper. Biol. & Med.*, **42**: December 1939.

⁹ Cokkinis, A. J., and McElligott, G. L. M., *Lancet*, **2**: 355-362, 1938.

¹⁰ Huntington, R. W., Jr.: *Proc. Soc. Exper. Biol. and Med.*, **38**: 328-331, 1938.

¹¹ Keefer, C. S., and Rantz, L. A., *Am. J. Syph., Gonorr. and Ven. Dis.*, **22**: 679-690, 1938.

¹² Kemp, H. A., *Texas State Jr. Med.*, **34**: 208-211, 1938.

¹³ Lockwood, J. S., Coburn, A. F. and Stokinger, H. E., *J. A. M. A.*, **111**: 2259-2264, 1938.

where active immunity develops in the presence of drug therapy. This is clinical proof that these drugs do not inhibit the development of immune mechanisms, a factor of importance in relation to some recently advanced theories of the sulfonamide therapy of gonococcus infections. Nor is there evidence that they stimulate antibacterial immunity which seems to be a function of the host aided only by less rapidly multiplying destructive forces. A disturbance of bacterial metabolism is advanced^{13 14 15} to explain this effect. Should this prove to be the mechanism of action, it is apparent from the wide number of organisms affected that the bacterial process altered must be a basic one and common to many bacteria. Furthermore, the clinical proof that sulfapyridine has proven so much superior against pneumococcus and sulfathiazole against the staphylococcus, indicates individual organism susceptibility. This should provide us with the greatest hope for the future development of compounds effective against the so far resistant virus, rickettsial and bacillary infections.

The introduction of new and effective compounds necessitates revision of our previous remarks on sulfanilamide therapy in various disease conditions. For our purpose this discussion can best be presented individually for the various diseases or disease groups.

BETA-HEMOLYTIC STREPTOCOCCUS INFECTIONS

The outstanding achievement obtained with sulfanilamide therapy in this group has not been surpassed in any other disease. Sulfanilamide remains the drug of choice.

Puerperal sepsis.—The infective process is limited, its spread and the invasion of the blood stream is prevented, hospitalization is shortened and the mortality markedly reduced. In cases with bacteremia, with or without peritonitis or extensive thrombophlebitis, mortality is reduced from around 70 percent to 40 percent.

Erysipelas.—There is sudden defervescence, arrest of the exanthematous process, and marked improvement in the general condition. The importance of early therapy is indicated by the observations of Rantz and Keefer.¹⁶ In cases receiving the drug before the third day of illness the total febrile course averaged 5.2 days with an average of 3 days after beginning treatment. In the cases first treated after the third day fever persisted for an average of 7.1 days and subsided in an average of 4.2 days after beginning medication. A mortality rate of 1.5 percent in adults and 12.9 percent in children has been

¹³ Garrod, L. P.: Chemotherapy of bacterial infections, *Lancet*, 1: 1125-1129; 1178-1182, 1938.

¹⁴ Lockwood, J. S.: Studies on the mechanism of the action of sulfanilamide: 111. Effect of sulfanilamide in serum and blood on hemolytic streptococci in vitro, *Jr. Immunol.*, 36: 155-193, 1938.

¹⁶ Rantz, L. A., and Keefer, C. S.: Sulfanilamide in treatment of erysipelas, *New Eng. J. of Med.*, 221: 809, November 23, 1939.

reported¹⁷ and compared to that of 9.2 percent in adults and 37.5 percent in children when erysipelas antitoxin was used.

The favorable comparison persists for patients treated with serum, ultraviolet rays, x-rays, and local treatment in which the group mortality was 14 percent for adults and 19.4 percent for children. However, Dick¹⁸ feels that sulfanilamide alone is inferior to x-ray alone. This disease is notorious for its tendency to recur and relapses and recurrences are noted¹⁸ as frequently in drug treated cases as among others. However, complications are said to occur less frequently.

Pharyngitis and tonsillitis.—Many workers have reported favorable results. This subject was recently reviewed by Rhoads and Afrenow,¹⁹ who reported a controlled series of cases and concluded that the value of this treatment in the average uncomplicated case of tonsillitis or pharyngitis due to hemolytic streptococci was questionable. They considered complications such as severe cervical adenitis, paranasal sinusitis, otitis media, mastoiditis, or meningitis to be indications for drug therapy. We must remember that a number of organisms cause sore throats and many of these conditions will, at times, simulate streptococcic pharyngitis. Certainly one must not overlook the importance of smear and culture, searching, especially in children, for a diphtheritic throat, and in adults for a Vincent's throat or a secondary syphilis. We have recently observed in a training station, a localized epidemic of acute upper respiratory infection of low attack rate, prolonged duration, and variable site of major involvement, from week to week. This condition has behaved clinically like a streptococcus infection, but we have been unable to isolate any particular organism consistently. The complication rate has been high but not of serious nature.

It is my feeling that sulfanilamide, while not curative, has reduced the incidence of complications and lessened morbidity in these cases. This group warrants careful differential diagnosis and in the absence of such conditions as diphtheria, secondary syphilis, Vincent's infection, acute leukemia, or agranulocytosis it is believed the low cost and harmlessness of sulfanilamide when given in moderate dosage for 5 to 7 days warrant its use. It is contemplated, in case of mobilization, that the sulfonamides will be used for their prophylactic as well as curative value in the control of respiratory infections.²⁰

Streptococcal pneumonia, bronchitis, pleurisy, and empyema.—Sulfanilamide is of great value in these conditions and especially in pneumonia and empyema it should be given early and in high dosage.

¹⁷ Nelson John, Rinzler, Harvey; and Kesley, M. P.: Sulfanilamide treatment in erysipelas, J. A. M. A., 112: 1044-1045, March 18, 1939.

¹⁸ Dick, George F., Year Book General Med., P. 115, 1939.

¹⁹ Rhoads, Paul S., and Afrenow, M. L.: Sulfanilamide in treatment of sore throat due to hemolytic streptococci, J. A. M. A., 114: 942-943, March 16, 1940.

²⁰ Hillman, Charles C.: Medical problems encountered in military service, Annals of Int. Med., 13: 2205-2212, June 1940.

Streptococcal peritonitis, osteomyelitis, and arthritis are other conditions in which this therapy is indicated. In some clinics sulfanilamide has been used in the treatment of acute hemorrhagic nephritis following a known *beta* hemolytic streptococcus infection and some benefit is thought to ensue.

Streptococci and war wounds.—The hemolytic streptococcus was responsible for about 70 percent of all the deaths due to infections of wounds in the first World War ²¹ and gave rise to most of the complications such as erysipelas, cellulitis, and septicemia. Stokes and Tyler found about 15 percent of wounds already infected by hemolytic streptococci on admission to the casualty clearing station (about 12 hours after injury), and a much larger proportion, 90 percent in one hospital, were found infected after a few days at the base. This suggests that sulfanilamide should be administered prophylactically over a period of at least 4 days during which time the reparative processes will have localized or rendered less serious any infection present.

The success attained in primary closure of badly contaminated compound fractures after débridement and topical application of sulfanilamide crystals offers great encouragement for the similar treatment of war wounds.²¹

Use in surgery.—Ravdin and his associates ²² have used sulfanilamide in acute appendicitis with associated peritonitis or abscess since 1936. They consider it of value as an accessory in reducing the mortality in such cases. The drug has been widely used by topical application in compound fractures and similar surgical emergencies with apparently favorable results.

The question of autoclaving crystalline sulfanilamide for use in open wounds has arisen. Available reports do not mention reactions or infections from the use of sulfanilamide powder.²³ There are no reports on the chemical changes occurring in crystalline sulfanilamide after autoclaving. However, it does tend to change color and the discolored product is not considered usable.

PNEUMOCOCCUS INFECTION

In the treatment of these infections, of which the most important is lobar pneumonia, sulfapyridine is far superior to sulfanilamide. It is effective in all types of pneumococcus infection, appreciably lowers the case fatality rate in bacteremic as well as nonbacteremic cases, and brings about a rapid decline in the patient's temperature and pulse rate in most cases. It is relatively cheap, does not deteriorate, is

²¹ Colebrook, Leonard: Chemotherapy of war wounds, *Foreign Letters, J. A. M. A.*, 114: 1683, April 27, 1940.

²² Ravdin, I. S., Lockwood, J. S., and Rhoads, J. E.: Use of sulfanilamide in treatment of peritonitis of appendiceal origin, *Penna. Med. J.*, 43: 1100-1104, May 1940.

²³ Queries and Minor Notes, *J. A. M. A.*, 115: 635, Aug. 24, 1940.

easy to store and administer, and has a wider range of usefulness than specific serotherapy. These features are especially adaptable to military medicine. However, the specific antipneumococcus sera are highly effective and, used with chemotherapy, shorten the period of illness.²⁴ While the use of sera entails accurate typing and preliminary serum-sensitivity testing, their value is being extended with the introduction of rabbit sera and the inclusion of more types of the pneumococcus. Many cases of pneumonia respond adequately to sulfapyridine which is cheaper and simpler to use than serotherapy. The respective indications for chemotherapy and serum therapy are still being studied. Finland²⁴ and co-workers recommended for the treatment of all acute pneumonias that—

1. Each case receive adequate bacteriologic control, including sputum examination with smear and typing and a blood culture before instituting treatment.
2. A complete blood count, urinalysis and blood N. P. N. before treatment and frequently thereafter when sulfapyridine is used.
3. Institution of sulfapyridine treatment in all cases as soon as pneumonia is diagnosed.
4. In adult patients over 40 years of age and in all pregnant or recently parturient women infected with the common pneumococcus types, specific serotherapy should be started as early as possible.
5. Serum should be given in all cases when there is no definite improvement after 24 to 36 hours of sulfapyridine therapy.^{25 26}
6. Cecil considers bacteremia in any type of pneumonia an indication for serum therapy.
7. It should be given to patients with pneumonia due to types II, III, and possibly V.²⁷
8. Other indications for the use of serum include those cases first seen late in the disease, debilitated individuals, involvement of two or more lobes, extension of the process under chemotherapy, and when sulfapyridine must be given cautiously as in renal disease, jaundice, granulocytopenia or severe anemia. The drug induces nausea and vomiting in 10 to 15 percent of cases. If necessary the sodium salt of sulfapyridine may be used intravenously,^{28 29} and is valuable in quickly obtaining a high blood level of the drug in seriously ill cases or when excessive vomiting occurs. The solution is highly alkaline and must be given cautiously. It produces sloughing if given outside the vein, and rigor at times.

The drug is rapidly excreted and must be given at 4-hour intervals day and night. Certain cases will improve on low blood levels not exceeding 2 mg. per-

²⁴ Finland, Maxwell; Spring, W. C., Jr.; Lowell, F. C. and Brown, J. B.: Specific serotherapy and chemotherapy of the pneumococcus pneumonias, *Ann. Int. Med.*, **12**: 1816-1828, May 1939.

²⁵ Finland, M., Spring, Wm. C., Jr., and Lowell, F. C.: Specific treatment of pneumococcal pneumonias, *Ann. Int. Med.*, **13**: 1567-1594, March 1940.

²⁶ Abernathy, T. J., Dowling, H. F. and Hartman, C. R.: Treatment of lobar pneumonia with sulfapyridine and sodium sulfapyridine with observation upon effective blood levels, *Ann. Int. Med.*, **13**: 1121-1138, January 1940.

²⁷ Finland, M.; Lowell, F. C. and Spring, W. C., Jr.: Clinical and laboratory studies on use of serum and sulfapyridine in treatment of pneumococcal pneumonias, *New. Eng. J. of Med.*, **272**: 739-748, May 2, 1940.

²⁸ Marshal, E. K., Jr., and Long, P. H.: Intravenous use of sodium sulfapyridine, *Jr. Am. Med. Assoc.*, **112**: 1672, 1939.

²⁹ Finland, Maxwell; Lowell, F. C.; Spring, W. C., Jr., and Taylor, T. H. L.: Parenteral sulfapyridine, *Ann. Int. Med.*, **13**: 1105-1121, January 1940.

cent.³⁰ During the first 3 to 4 days a blood concentration ³¹ of 4 to 6 mg. percent,³² or of 6 mg. percent or above,²⁶ are considered optimum. Whereas 90 percent of patients with mean blood concentrations above 6 mg. percent recovered by crisis, only 66 percent of those with levels below 6 mg. percent recovered in this manner.²⁶ The dosage used by various clinicians is listed in table 1.

TABLE 1.—*Dosage used by various clinicians.*

Reference	Initial oral dose in grams	Further dosage in grams	Average total dose in grams
33	2	1 q. 4. h.	33.
34	2 and then repeat in 2 hours.	1 q. 4. h. until temp. normal 24 hours. Then reduce to 1 gm. q. 6. h. for 24 hrs. Then 1 gm. t. i. d. for 24 hrs.	20-25.
35	3	2 gms. in 2 hrs. Then 1 gm. q. 4. h.	15-25.
30	2	1 gm. q. 4. h.	25.
36	2	1 or 2 gms. 4 hrs. later. Then 1 gm. q. 4. h. until temp. is normal. Then 0.5 gm. q. 4. h.	15-25.
37	2	1 gm. q. 4. h. for 12 dos. and then 1 gm. q. 6. h.	25.
26	4	1 gm. q. 4. h. until temp. normal for 48-72 hrs. Then 1 gm. q. 6. h. for 48-72 hrs. and then 0.5 gm. q. 4. h. as needed.	
38	4	1 gm. q. 4. h. until temp. normal for 48 hrs. Then 1 gm. q. 6. h. until resolution well advanced. Then 0.5 gm. 4 x daily until pt. ready to leave his bed.	

As a rule the sulfapyridine treated cases show a prompt drop in the temperature within 18 to 48 hours. The signs of consolidation persist until the time of the expected crisis. If the apparent recovery leads to discontinuance of therapy before resolution, a relapse is likely to occur. The drug has no appreciable effect on the rate of development, the quantity or quality of immune bodies,³⁹ and final recovery results from the development of active immunity or the administration of immune serum. In nearly 80 percent of drug treated cases recovery

³⁰ Pepper, D. C., Flippin, H. F., Schwartz, L. and Lockwood, J. S.: Results of sulfapyridine therapy in 400 cases of typed pneumococcal pneumonia, *Am. J. M. Sc.*, **198**: 22-35, July 1939.

³¹ Marshal, E. K., Jr., *J. Biol. Chem.*, **128**: 537, May 1939. (This revised procedure is more sensitive and more easily performed than the method formerly described by Marshal.)

³² Long, P. H. and Bliss, E. A.: Clinical and experimental use of sulfanilamide, sulfapyridine and allied compounds, 1939, The MacMillan Company, New York, 230-231.

³³ Ruegsegger, J. M.; Hamburger, M., and Cockrell, Sarah L.: Comparative use of sulfapyridine and specific serum in pneumococcal pneumonia, *Ohio State Med. J.*, **36**: 257-262, March 1940.

³⁴ Whitby, dosage originally recommended.

³⁵ Smith, F. J. and Needles, R. J.: Report of 50 cases of acute lobar pneumonia in adults treated with sulfapyridine, *Am. J. M. Sc.*, **198**: 19-21, July 1939.

³⁶ Cecil, Russell L.: Editorial on pneumonia, *Digest of Treatment*, **3**: 440-441, December 1939.

³⁷ Williams, R. H. and Morgan, H. J.: Treatment with sulfapyridine of 50 patients with pneumococcal lobar pneumonia, *Southern Med. J.*, **32**: 608-613, June 1939.

³⁸ Long, P. H., and Feinstone, W. H.: Observations upon experimental and clinical use of sulfapyridine II. Treatment of pneumococcal pneumonia with sulfapyridine, *Ann. Int. Med.*, **13**: 487-512, September 1939.

³⁹ McIntosh, J., and Whitby, L. E. H.: Mode of action of drugs of sulphonamide group, *Lancet*, **1**: 431-435, February 25, 1939.

occurs without the appearance of an excess of type specific antibodies ⁴⁰ as determined by the precipitin reaction with the specific polysaccharide. This low antibody content may account for recrudescences with discontinuance of sulfapyridine therapy.

The mortality rate in pneumococcus pneumonia is influenced by so many factors that no brief discussion can adequately present the subject. It may be stated that the results with sulfapyridine compare very favorably with those from the use of specific sera. A rough estimate of comparative results is presented in table 2.

TABLE 2.—*Mortality percent with various forms of treatment*

Reference	No. of cases	Nonspecific	Sulfa-pyridine	Specific serum	Serum and sulfapyridine
41.-----	5, 977	30. 83	10. 69	17. 84	14. 67
	5, 554	-----	-----	15. 79	-----
	3, 608	-----	6. 26	-----	-----
25.-----	211	-----	-----	13. 3	-----
	129	-----	-----	-----	24. 0
	225	-----	17. 8	-----	-----
	472	28. 6	-----	-----	-----
36.-----	956	-----	7. 1	-----	-----

It is the most seriously ill patient who receives both serum and sulfapyridine and this probably accounts for the increased mortality in the group receiving both agents.

Recent experience ^{6 42} indicates that sulfathiazole is equally as effective as sulfapyridine against pneumococcus infections. It is used in the same dosage, nausea and vomiting appear slightly less troublesome and for rapid administration a 5-percent solution of sodium sulfathiazole may be given intravenously. One dose of 0.06 gm. per kilo raises the free sulfathiazole blood serum content to 8 to 10 mg. percent.

MENINGITIS

The sulfonamides are of undoubted value in these infections. The controversial issues concern the respective value of chemotherapy and specific serum and antitoxin, or their combined use, and the indications for spinal puncture.

In a series ⁴³ of 65 cases of meningococcus meningitis, treated with

⁴⁰ Kneeland, Y., Jr., and Mulliken, B.: Antibody formation in cases of lobar pneumonia treated with sulfapyridine, *J. Clin. Invest.*, **19**: 307, March 1940.

⁴¹ Faller, C. P.; Quickel, K. E. and Smith, C. W.: Statistical study of 5, 977 cases of pneumonia, with mortality statistics of 9,162 serum and sulfapyridine treated cases collected from recent literature, *Penna. Med. J.*, **43**: 789-801, March 1940.

⁴² Carey, B. W.: Use of sulfanilamide and related compounds in diseases of infancy and childhood. Address to General Scientific Meeting, A. M. A. June 11, 1940.

⁴³ Banks, H. Stanley: Chemotherapy of meningococcal meningitis, *Lancet*, **237**: 921, October 1939.

intensive serum intravenously and sulfanilamide in varying dosage, the mortality was 12.3 percent.

These figures are exclusive of six cases in which treatment could not be applied and four of which died, thus giving a gross mortality of 16.9 percent. There were 13 complications and 3 sequels (deafness). Of 72 cases, comparable in severity to this group, which were treated with sulfanilamide and sulfapyridine, the fatality rate was 1.4 percent. An additional four cases were either untreated or inadequately treated, and all died, giving a gross mortality of 6.6 percent. In this group there were six complications and two sequels (deafness).

In another series ⁴⁴ the fatality rate in the adequately treated cases was 12 percent (sulfanilamide) and 13 percent (sera and sulfanilamide).

The recovery of 3 of 6 cases of streptococcus viridans meningitis following sulfapyridine therapy is reported.⁴⁵

Sappington and Favorite ⁴⁶ reviewed the use of sulfanilamide in the various bacteriological types of meningitis. They feel the use of specific sera with chemotherapy is of unproven value except in those cases due to the *H. influenzae*. They collected 25 cases of influenzal meningitis which were treated with sulfanilamide, with or without serum. The mortality was 96 percent as compared to a mortality of 84.6 percent in 201 serum treated cases. Silverthorne ⁴⁷ and Carey ⁴² also recommend the use of serum in these cases.

There seems to be a tendency ⁴⁷ in diseases like meningococcus meningitis and tetanus to do away with or minimize intraspinal medication, ⁴⁸ and frequent spinal taps ^{49 44} with improving results. The Ministry of Health (London) ⁵⁰ advocates an initial lumbar puncture for diagnosis and further punctures only to relieve pressure symptoms (intense headache) and on alternate days to verify the efficacy of chemotherapy. Lang ⁵¹ emphasizes the value of antitoxin, presents a scheme of sulfanilamide therapy, and points out that spinal taps were used frequently for relieving pressure and removing organisms.

In the treatment of meningitis it is to be remembered that sulfanilamide and sulfapyridine are equally effective against the streptococcus and the meningococcus.⁵⁰ The pyridine derivative is superior against the pneumococcus and the gonococcus. Early administration

⁴⁴ Waghelstein, J. M.: Sulfanilamide in treatment of 106 patients with meningococcal infections, J. A. M. A. 111: 2172, 1938.

⁴⁵ Mitchell, Wm. J.; Bower, A. C. and Hamilton, P. M.: Use of sulfapyridine in streptococcus viridans meningitis, Am. J. Med. Sc., 200: 75-77, July, 1940.

⁴⁶ Sappington, S. W. and Favorite, G. O.: Sulfanilamide and meningitis, Ann. of Int. Med., 13: 576-598 October 1939.

⁴⁷ Silverthorne, N., et al: Influenzal meningitis, J. of Pediat., 16: 456-462 April 1940.

⁴⁸ Hoyne, A. L.: Meningococcus meningitis, Jour. Pediat., 11: 863, 1937.

⁴⁹ Toomey, J. A., and Kimball, E. R., Jr.: Meningitis caused by streptococcus haemolyticus and treated with sulfanilamide, Jr. Am. Med. Assoc., 112: 2586, June 24, 1939.

⁵⁰ Foreign Letters, J. A. M. A. 114: 1820, May 4, 1940.

⁵¹ Lang, Fred: Meningococcus antitoxin in cerebrospinal fever, U. S. Nav. Med. Bull., 38: 52-72, January 1940.

is imperative. With the finding of cloudy spinal fluid sulfapyridine should be given immediately. If the organism proves to be the streptococcus or the meningococcus then the cheaper and less toxic sulfanilamide can be substituted. The intensive use of intravenous serum therapy is not considered compensable for low dosage of sulfapyridine.⁴³ However, for some strains of meningococci, serum therapy has been more successful; for others the drugs have been better.⁵² The combination of drug and serum therapy, at least experimentally, apparently gives results far better than with either agent alone.

The drugs should be given every 4 hours, day and night. The concentration of sulfapyridine in the cerebrospinal fluid should be maintained at 5 mg. per 100 cc. for 3 days and a diminishing concentration for a further 5 to 6 days.^{43 50} The drug treatment should be completed in from 7 to 9 days in order to avoid the more dangerous toxic effects. The dosage for infants is proportionately higher than for adults. Infants easily tolerate 3 grains of sulfapyridine daily for the first 3 days.⁵⁰

The use of sulfathiazole is not recommended ⁴² because it apparently is not excreted in the spinal fluid in sufficient quantity to justify its use. However, a case of *S. aureus* meningitis incident to an upper lip furuncle, was successfully treated with sulfathiazole.⁵³ This 20-month old child also received daily spinal punctures and numerous blood transfusions.

OTITIC AND MASTOID INFECTIONS

A review of the use of sulfanilamide in otolaryngology was presented by Schenck ⁵⁴ who concluded that most observers report favorable responses to the drug but that the carrier state was usually not much affected. A series of 621 cases of suppurative otitis media ⁵⁵ were treated with sulfanilamide and then sulfapyridine when pneumococci were isolated. The incidence of mastoiditis was 3.4 percent as compared with 22.7 percent before the introduction of sulfanilamide. The importance of isolating the causative organism, as a guide to which sulfonamide product to use, is indicated by a recent bacteriological study.⁵⁶ The percentage incidence of the various organisms is listed in table 3.

⁴² Branham, S. E.: Pub. Health Rep., 55: 12-25, January 5, 1940.

⁴³ Dietel, F. W. and Kaiser, A. D.: J. A. M. A. 115:601, August 1940.

⁴⁴ Schenck, H. P.: Use of sulfanilamide in otolaryngology, Arch. Otolaryng., 28: 698, November 1938.

⁴⁵ Horan, V. G. and French, S. G.: Prevention of mastoiditis; survey of 621 cases of acute otitis media treated with sulfanilamide, Lancet, 1: 680-683, April, 1940.

⁴⁶ Fowler, E. P. Jr.: Otitis media and its extensions, Bull. New York Acad. Med., 16: 24, January 1940.

TABLE 3.—*Incidence of organism and sulfonamide recommended*

Type of organism	Percent incidence	Sulfonamide
Staphylococcus.....	35 plus.....	Sulfathiazole.
Hemolytic streptococcus.....	30.....	Sulfanilamide.
Pneumococcus.....	12 to 16.....	Sulfapyridine.
Other organisms not susceptible to sulfonamides.	15 plus.....	

In a bacteriologic study of acute mastoiditis cases coming to operation, Bowers⁵⁷ found *S. hemolyticus* in 52 percent, nonhemolytic streptococcus in 10 percent, pneumococcus type III in 5 percent, staphylococcus in 15 percent and no growth in 18 percent.

In the successfully treated cases dramatic subsidence of symptoms occurs⁵⁶ in 3 to 4 days. In most cases no pain or fever was noted after 2 days of therapy. This suggests that if there is no effect in 3 to 4 days, chemotherapy is not effective and surgery is more apt to be required. When surgery is necessary the adjuvant use of chemotherapy is indicated as an aid in preventing meningitic extension. It would seem that these drugs greatly reduce the incidence of mastoiditis and allow a more conservative approach to the problem. In this connection Bowers⁵⁷ has reported a decrease from 24 to 11 percent in the number of cases of acute purulent otitis media requiring mastoid operation when adequate chemotherapy is instituted early in the course of the infection. The drug must be continued for at least 1 week after the disappearance of all aural symptoms in order to prevent relapse.

LYMPHOCYTIC CHORIOMENINGITIS

One case was reported⁵⁸ which had four recrudescences over a period of 4 months. The use of sulfanilamide gave apparent success.

SCARLET FEVER

Silverman⁵⁹ has recalled the striking change in the character of scarlet fever over the last 75 years, the mortality in 1938 being one one-hundredth of that in 1861 which was 100 per 100,000. This increasing mildness adds to the difficulty in assessing the therapeutic effects of serum and sulfanilamide. There is no evident influence on the toxic phase and chemotherapy does not replace specific serum and antitoxin. In a recently reported series of 300 cases⁶⁰ there was an apparent reduction in the number of septic complications and when

⁵⁷ Bowers, W. C.: Observations on 793 cases of acute purulent otitis media, *J. A. M. A.*, 115: 178-181, July 20, 1940.

⁵⁸ Leichenger, J.; Milzer, A., and Lack, H.: Recurrent lymphocytic choriomeningitis treated with sulfanilamide, *J. A. M. A.*, 115: 436-440, August 10, 1940.

⁵⁹ Silverman, A. C.: Sulfanilamide in treatment of scarlet fever, *New York State J. of Med.* 40: 317-326, March 1940.

⁶⁰ Bozalis, G. S. and Barnett, H. L.: Sulfanilamide in treatment of scarlet fever, *Mo. State Med. Assn. J.*, 37: 137-142, April 1940.

complications did occur a prompt response to sulfanilamide treatment took place. This has been amply confirmed ^{61 62 63}. Carey ⁴⁸ feels that the combined use of serum and chemotherapy has the best effect and this seems to represent the concensus of opinion.

RHEUMATIC FEVER

Rheumatic children were given ⁶⁴ maintenance doses of sulfanilamide (about 2 gm. daily) over a period of months and observed with respect to the incidence of hemolytic streptococcus infections and recurrences of rheumatic fever. No toxic effects from the drug were observed. Only 1 of the 26 patients so treated contracted an infection with hemolytic streptococci in the throat flora and only 1 of the 26 highly susceptible rheumatic children developed active rheumatism. However, sulfanilamide administered to rheumatic subjects after the onset of streptococcic throat infections did not prevent rheumatic recrudescences.

This is considered to be an important contribution to the prophylaxis of rheumatic fever ⁶⁵ and seems to offer real promise as a practical means of preventing recurring attacks of the disease.⁶⁶

ATROPHIC ARTHRITIS

The use of sulfanilamide in this condition has been extensively studied. Coggeshall and Bauer ⁶⁷ found it without beneficial effect on either the clinical course or the sedimentation rate. The results of others,^{68 69} have been equally disappointing, Margolis reporting that 44 of 92 physicians who answered a questionnaire were almost unanimous in condemning the use of sulfanilamide in this disease.

CHRONIC ULCERATIVE COLITIS

This disease, of unknown etiology, is characterized by spontaneous exacerbations and remissions. There is no known specific remedy. The prompt improvement occurring so uniformly with the use of

⁶¹ Benn, E. C.: Sulfanilamide in treatment of scarlet fever, *Brit. Med. J.*, 2: 644, September 23, 1939.

⁶² Hamilton, P. M. and Togasaki, Y.: *J. Pediat.*, 14: 655-657, May, 1939.

⁶³ Long, P. H., Haviland, J. W., Edwards, L. B., and Bliss, E. A.: Toxic manifestations of sulfanilamide and its derivatives, *J. A. M. A.*, 115: 364-368, August 3, 1940.

⁶⁴ Coburn, A. F.; and Moore, L. V.: Prophylactic use of sulfanilamide in streptococcal respiratory infections, with especial reference to rheumatic fever, *J. Clin. Invest.*, 18: 147-155, Jan. 1939.

⁶⁵ Cecil, Russell, L.: Therapy of rheumatic fever, *J. A. M. A.*, 114: 1443-1447, April, 13, 1940.

⁶⁶ Thomas, C. B., and France, Richard: A preliminary report of the prophylactic use of sulfanilamide in patients susceptible to rheumatic fever, *Bull. Johns Hopkins Hosp.*, 114: 67-77, 1939.

⁶⁷ Coggeshall, H. C. and Bauer, Walter: Treatment of gonorrheal and rheumatoid arthritis with sulfanilamide, *New Eng. J. Med.*, 220: 85, January 19, 1939.

⁶⁸ Margolis, H. M. and Eisenstein, V. W.: Some specific measures in the treatment of rheumatoid arthritis, *J. A. M. A.*, 114: 1429-1433, April 13, 1940.

⁶⁹ Bethes, Oscar W.: Atrophic arthritis, *Internat. Med. Digest*, 36: 172-175, March 1940.

Neoprontosil (oral) was thought to justify a thorough trial.⁷⁰ This relatively nontoxic drug was used in dosage of from 4 to 5½ grams daily for 10 to 14 days. Many cases received an additional 2½ grams daily for an additional 10 to 14 days, followed by a 7- to 14-day rest period. In general this program was followed for 3 months, with further administration, in reduced dosage, where indicated. Of 29 patients 44.8 percent were considered to have excellent results, 44.8 percent fair results, and only 10 percent had poor results. However, Bargaen⁷¹ still considers this therapy to be in a trial stage.

TULAREMIA

There is no accurate data available, but unpublished information on its use in 30 cases indicates little or no favorable action of the drug.⁷² One case has been reported⁷³ which was successfully treated with serum and sulfanilamide.

PLAGUE

A possible beneficial action of Prontosil was noted by Carmen.⁷⁴ It is now reported⁷⁵ that sulfathiazole, and to a lesser degree sulfapyridine, are effective for plague in mice. It is recalled that in human plague spontaneous recovery invariably results if the lymph glands prevent the spread of infection to the blood stream. The occurrence of such cases in different outbreaks is from 20 to 50 percent. If a septicemia develops, death invariably follows. Since these drugs protect mice even after septicemia has developed it is to be hoped that they will prove even more effective in the less susceptible human.

SUBACUTE BACTERIAL ENDOCARDITIS

The use of Prontosil, sulfanilamide and sulfapyridine,^{76 77} of heparin and sulfapyridine,⁷⁸ and of fever therapy and sulfanilamide⁷⁹ have been on the whole unsuccessful. The drug may lower the temperature, sterilize the blood stream, and improve the patient's well-being, but these effects have been almost uniformly temporary.⁸⁰

⁷⁰ Brown, A. E.; Herrell, W. E., and Bargaen, J. A.: Neoprontosil (oral) in the treatment of chronic ulcerative colitis, *Ann. Int. Med.*, 13: 700-714, October 13, 1939.

⁷¹ Schlicke, C. P., and Bargaen, J. A.: Fulminating ulcerative colitis, *Minn. Med.*, 23: 348 May 1940.

⁷² Queries and minor notes, *J. A. M. A.*, 114: 1007, March 16, 1940.

⁷³ Powers, G. L., and Powers, E. G.: Tularemia, report of a case treated with sulfanilamide and anti-serum, *Tex. State J. Med.*, 35: 350, September 1939.

⁷⁴ Carmen, J. A.: *East African Med. J.*, 14: 362, February 1938.

⁷⁵ Sokhey, S. C., and Dikshit, B. B.: Sulfathiazole in bubonic plague, *Lancet*, 1: 1040, June 8, 1940.

⁷⁶ Major, Ralph H.: Effect of sulfanilamide compounds on endocarditis, *Am. J. Med. Sc.*, 199: 759-768, June 1940.

⁷⁷ Spink, W. W., and Crago, F. H.: *Arch. Int. Med.*, 64: 228, 1939.

⁷⁸ Kelson, S. R., and White, Paul D.: *J. A. M. A.*, 113: 1700-1702, November 4, 1939.

⁷⁹ Krusen, F. H., and Bennett, R. L.: *Proc. Staff Meet. Mayo Clinic*, 15: 328-333, May 22, 1940.

⁸⁰ Steele, H. H.: *New Eng. J. of Med.*, 222: 1067-1071, June 27, 1940.

MALARIA

The use of sulfonamide compounds in human malaria cannot be recommended until much further carefully controlled experimental evidence is available on human malarial plasmodia⁸¹

TRACHOMA

It is generally recognized that sulfonamide therapy is beneficial in trachoma. In reporting on their cases Richards, Forster, and Thygeson⁸² noted striking improvement. In the first week corneal subepithelial infiltrations disappeared and the caliber of pannus vessels and number of fluoresin-staining epithelial lesions decreased. In about 2 weeks conjunctival secretion diminished and the membrane became thinner and paler. The follicles disappeared slowly. Four and one-half months after beginning treatment all conjunctivas were smooth and follicle-free. The disappearance of trachoma virus was indicated by repeated failure to demonstrate inclusion bodies during treatment and pooled epithelial scrapings failed to infect baboons. The corneal complications, pannus, keratitis, and ulcers seem to respond best^{83 84} and corneal complications were rarely observed after treatment with sulfanilamide. Irrespective of the type of trachoma it was noted⁸⁵ that the condition was rendered asymptomatic in roughly 20 percent, improved in 40 percent and unchanged in 40 percent. Furthermore it was held that supplemental chemical and mechanical measures were necessary. Spining⁸⁶ reports that tearing, photophobia and bulbar conjunctivitis were improved or had disappeared after 3 to 4 days of treatment. The conjunctivas became less congested and granulations appeared paler and smaller. However, in his opinion, the results were no more striking than would be expected in similar cases hospitalized for the same period and given any of the usual forms of treatment. A check up 3 months later showed considerable improvement as compared with cases treated by other methods. Spearman and Vandever⁸⁷ treated 2 cases with sulfapyridine and noted remission of pathologic signs and improvement of vision after being refractory to other treatment.

⁸¹ Reed, A. C.: Treatment of malaria, *J. A. M. A.*, **115**: 603, August 24, 1940.

⁸² Richards, Polk; Forster, W. G., and Thygeson, P.: Treatment of trachoma with sulfanilamide, *Arch. Ophthalmol.*, p. 577-580, April 21, 1939.

⁸³ Sie-Boen-Lian: Sulfanilamide therapy of trachoma, *Ophthalmologica*, Basel, **98**: 208 December 1939. *Abst. J. A. M. A.*, **114**: 1497, April 13, 1940.

⁸⁴ Lee, Otis S., Jr. and Rottenstein, Hans: Trachoma treated with sulfanilamide and its derivatives, *J. A. M. A.*, **115**: 107-112, July 13, 1940.

⁸⁵ Julianelle, T. A., et al: Effect of sulfanilamide on course of trachoma, *Amer. J. Ophthalmol.*, **22**: 1244, November 1939.

⁸⁶ Spining, W. D.: Some observations on use of sulfanilamide in trachoma and associated ocular conditions, *Am. J. Ophthalmol.*, **23**: 271-275, March 1940.

⁸⁷ Spearman, M. P. and Vandever, W. E.: Sulfapyridine in trachoma, *J. A. M. A.*, **113**: 1807, November 11, 1939.

STAPHYLOCOCCUS INFECTIONS

The introduction of the thiazole product has brought forth spectacular reports of its efficacy in septicemia^{88 89 90 91}, staphylococcus arthritis and perinephritic abscesses,⁹¹ osteomyelitis and carbuncles.⁹² In the latter two conditions surgery is often necessary.

Butler, however, has found sulfathiazole of little or no effect in a staphylococcal infection when the blood culture was positive.

Another interesting use of this drug has been its incorporation with bath soap for use in the nursery⁹¹. With its introduction impetigo disappeared only to return when the drug was discontinued.

POLIOMYELITIS

In experimental studies sulfanilamide^{93 94}, and sulfapyridine^{95 96} have failed to prevent the infection in monkeys. Rhett⁹⁷ used Neoprontosil during an epidemic of poliomyelitis with some apparent inhibitory effect and Wagner⁹⁸ thought sulfapyridine of some value.

MEASLES

Anderson⁹⁹ observed that sulfanilamide had little effect in shortening the duration of primary pyrexia or in hastening the cure of the major complications with the exception of bronchopneumonia the duration of which was materially less in cases receiving sulfanilamide. This therapy has been credited¹⁰⁰ with lowering the incidence of complicating bronchopneumonia from 4.8 to 1.7 percent. The mortality of such bronchopneumonia was lowered from 28.7 to 11.1 percent. It is credited with good results in prophylaxis and treatment of such complications as otitis, bronchopneumonia, adenitis and tonsillitis.

⁸⁸ Herrell, W. E. and Brown, A. E.: Clinical use of sulfamethylthiazol in infections caused by staphylococcus aureus, Proc. Staff Meet. Mayo Clinic, 14: 753-759, November 29, 1939

⁸⁹ Keillher, T. F. and Carlen, S.: Treatment of staphylococcal septicemia with sulfamethylthiazole, M. Ann. Dist. of Columbia, 9: 78, March 1940.

⁹⁰ Stirling, W. O.: Sulfathiazole: Two cases of septicemia with recovery, J. A. M. A., 115: 118-120, July 13, 1940.

⁹¹ Carroll, G. L. et al: Therapy of the thiazole compounds of sulfanilamide, Scientific Session, Section on Urology, A. M. A., June 12, 1940.

⁹² Foreign Letters, J. A. M. A., 115: 471, August 10, 1940.

⁹³ McKinley, E. B.; Acree, E. G., and Meck, I. S.: Sulfanilamide and virus diseases, Science, 87: 43-44, January 14, 1938.

⁹⁴ Kelson, S. R.: Use of sulfanilamide in experimental poliomyelitis, Proc. Soc. Exper. Biol. and Med., 36: 718-720, June 1937.

⁹⁵ Rosenow, E. C.: Failure of sulfapyridine to protect against experimental (virus) poliomyelitis, Proc. Staff Meet. Mayo Clinic, 14: 490-495, August 2, 1939.

⁹⁶ Toomey, J. A. and Takacs, W. S.: Sulfapyridine and experimental poliomyelitis, Arch. Pediat., 55: 384 June 1939.

⁹⁷ Rhett, W. M.: Prophylaxis and treatment of poliomyelitis with Neoprontosil, J. of Ped., March 1940.

⁹⁸ Wagner, J. C.: Anterior poliomyelitis treated with sulfapyridine, J. A. M. A., 112: 1934-1935 May 13, 1939.

⁹⁹ Anderson, T.: Sulfanilamide in treatment of measles, Brit. Med. Jour., 1: 716-718, April 1939.

¹⁰⁰ Thompson, A. R. and Greenfield, C. R. M.: Chemotherapy in measles and whooping cough, Lancet, 2: 991-994, October 29, 1938.

OTHER INFECTIOUS DISEASES

Sulfanilamide has been considered of value in preventing the pustulation and therefore scarring in smallpox.¹⁰¹ While there was no influence on the course of experimental pertussis in mice¹⁰² it is apparently of value in preventing and treating the bronchopneumonia which so often complicates pertussis.¹⁰¹ It is considered of value in impetigo contagiosa complicated by lymphadenopathy and fever.¹⁰⁸ Sulfanilamide was ineffective in freeing a paratyphoid A carrier of organisms¹⁰⁴ and the general opinion seems to credit sulfonamide therapy with little or no effect upon typhoid and paratyphoid organisms. Such treatment of bacillary infections in general is still in the experimental stage.¹⁰⁵

There is little evidence that sulfanilamide therapy produces any lasting effect in brucellosis¹⁰⁶ despite the favorable early reports.¹⁰⁷

In civil practice antitoxin and the roentgen ray are used extensively in therapy of gas gangrene. These are not ideal therapeutic agents for use on the battlefield. There is some evidence that sulfapyridine is more effective²¹ than sulfanilamide.¹⁰⁷

There have been favorable reports on the sulfonamide therapy of lupus erythematosus.^{108 109} Andrews' text carries the assertion it is of value in the acute disseminated forms.

TUBERCULOSIS

In addition to the references previously cited¹⁰⁷ other reports^{110 111} continue to indicate lack of any therapeutic effect. Its use has been advocated in connection with tuberculosis surgery for the purpose of inhibiting secondary infection.

GONOCOCCUS INFECTIONS

The use of sulfanilamide in these infections was previously reviewed for this publication. Dees and Young¹¹² and the Rheumatism Review committee,¹¹³ among others, have presented interesting reports on

¹⁰¹ McCammon, W. O.: J. A. M. A., 112: 1936-1937, May 13, 1939.

¹⁰² Cruickshank, J. C.: Sulfanilamide and M&B 693 in experimental pertussis in mice, Lancet: 2: 310-311, August 6, 1938.

¹⁰³ Diseases of the skin, Andrews, Second Edition, page 276.

¹⁰⁴ Saphir, Wm. and Howell, K. M.: J. A. M. A., 114: 1938, May 18, 1940.

¹⁰⁵ Queries: J. A. M. A., 115: 78, July 6, 1940.

¹⁰⁶ Queries and Minor Notes, J. A. M. A., 114: 1688, April 27, 1940.

¹⁰⁷ Bell, Robert A.: Sulfanilamide and chemotherapy, U. S. Nav. Med. Bull., 37: 73-112, January 1939.

¹⁰⁸ Weiner, A. L.: Disseminated lupus erythematosus treated with sulfanilamide, Arch. Dermat. and Syphil., 41: 534, March 1940.

¹⁰⁹ Barber, H. W.: Lupus erythematosus: effects of sulfonamides, Lancet, 1: 583, March 30, 1940.

¹¹⁰ Birkhaug, K. E.: Abstract J. A. M. A., 114: 1972, May 11, 1940.

¹¹¹ Frellich, E. B.; Coe, G. C. and Wien, N. A.: Use of sulfanilamide in pulmonary tuberculosis, Ann. Int. Med., 13: 1042-1046, December 1939.

¹¹² Dees, John E., and Young, Hugh H.: Sulfanilamide therapy in gonorrhea, Ven. Dis. Info., 20: 33-40, February 1939.

¹¹³ Sixth Rheumatism Review, Ann. Int. Med., 13: 1163-1699, March 1940.

this subject. Since that time there have arisen claims ¹¹⁴ that the administration of sulfanilamide should be delayed for a variable length of time after the appearance of symptoms. For instance, a delay of 20 days has been advocated ¹¹⁵ because the cure rate was found to increase from 74.5 percent, in the group treated before this time interval had elapsed, to 96.1 percent in cases having the disease for 3 weeks or longer before beginning therapy. The early and late relapse rate is said to be less under the delayed method of treatment.¹¹⁶

Many advocates of the delay system feel that it allows time for the development of immunity processes unhindered by chemotherapy. While there are no laboratory methods capable of detecting the immunologic factor in gonococcus infections ¹¹⁵ the specificity of the complement fixation test is not influenced by sulfanilamide therapy,^{117 118} From what is known of the action of these drugs in promoting cure of infectious diseases there is no evidence of interference with immunity development. For instance, it is generally held that recovery from pneumococcus pneumonia results from the development of active immunity at the time drug bacteriostasis is effective. The immunity to the gonococcus is a more variable and tardy development. The infection is self-limited, rarely causes death, and with the lapse of time cure will result in a high proportion of cases.

The principal reasons for early treatment are control of infectiousness, prevention of extension and complications, and relief of mental torture. The most striking action of the sulfonamides on gonococcus infections is the prevention of extension and complications. Furthermore approximately one-third of all cases will be cured in the first 10 days,¹⁰⁷ or 3 weeks ¹¹⁹ of therapy. It is believed with Cook ¹²⁰ that delayed treatment is of no benefit in relation to the defense mechanism. Furthermore, to mention a few, Cook,¹²⁰ King,¹²¹ Herrold ¹¹⁹ and others ¹¹⁸ feel there is no justification for withholding treatment once the diagnosis is made. Regardless of the merits of this controversy as it applies to sulfanilamide, it appears that sulfapyridine is equally effective in any stage of the disease.¹²²

The early statistics were not conclusive ¹¹² as to whether the use of local treatment is a valuable addition to chemotherapy. Some

¹¹⁴ Cokkinis, A. J., and McElligott, G. L. M.: *Lancet*, 2: 1264, 1938.

¹¹⁵ Van Slyke, C. J. and Mahoney, J. F.: Further observations in sulfanilamide therapy of gonococcal infections, *New York State J. Med.*, 40: 122, January 15, 1940.

¹¹⁶ Cokkinis, A. J., and McElligott, G. L. M.: Relapse after sulfonamide cure of gonorrhea, *Brit. Med. J.*, 2: 1080, December 2, 1939.

¹¹⁷ Cohn, Alfred: Gonococcus complement fixation test in gonococcus infections treated with sulfanilamide, *Am. J. Syph., Gonorr., and Ven. Dis.*, 23: 461, July 1939.

¹¹⁸ Keefer, Chester S. and Rantz, L. A.: Sulfanilamide in the treatment of gonococcal arthritis, *Am. J. Med. Sc.*, 197: 168-181, February 1939.

¹¹⁹ Herrold, R. D.: Chemotherapy in treatment of gonorrhea, Scientific Session, A. M. A., Section on Urology, June 12, 1940.

¹²⁰ Cook, E. N.: Chemotherapy in urology, General Scientific Meeting, A. M. A., June 11, 1940.

¹²¹ King, A. J.: Discussion of sulphonamide therapy in gonorrhea, *Brit. J. Ven. Dis.*, 15: 106, April 1939.

¹²² Foreign Letters, *J. A. M. A.*, 113: 2253, December 16, 1939.

workers¹²¹ have considered the results in no way inferior when irrigations are withheld. One clinic¹²² divided 100 untreated, anterior urethritis cases into four different treatment groups of 25 each.

The first received only sulfanilamide with an average duration of 39.1 days and no serious untoward reactions.

The second received the same amount of sulfanilamide plus anterior urethral injections of 5 percent silvol or 0.25 percent protargol with an average duration of 51 days and more reactions.

The third were given anterior urethral irrigations of 5 percent silvol preceded by irrigations of 1:8000 KMnO₄. The average duration was 51 days and complications were significantly higher.

The fourth group received daily anterior irrigations of a colloidal silver suspension (Colsargen) and the average duration was 46.9 days with 36 percent complications.

Contrary to such experiences there is a widespread support of the combined therapy. Cook¹²⁰ of the Mayo Clinic, considers local therapy definitely advantageous and states the detailed comparative reports by English clinicians bear this out. Speaking from a wide clinical experience, Pelouze¹²⁴ considers that chemotherapy plus local treatment gives the best results. The British Army has adopted the combined therapy as routine in gonococcus infections.¹²⁵ It is believed the use of local therapy has been well justified. Once the gonococcus becomes lodged in the prostate, a series of prostatic massages must be given in addition to chemotherapy.

The choice of sulfonamide compound is worthy of comment. It was pointed out previously¹⁰⁷ that sulfanilamide will effect cure in gonorrhea in about one-third of cases in 10 days, in another third in 1 month, and the final third require an average of 2 months. This is a conservative, and remains a fairly correct appraisal, of the value of this compound. Culture experiments¹²⁶ indicate that sulfapyridine is more toxic for the gonococcus than is sulfanilamide.

In clinical use it has been considered¹²¹ more potent against the gonococcus than any of its predecessors. The early relapse rate in sulfapyridine treated male patients¹¹⁶ was found to be 8.6 percent, or less than half that with sulfanilamide which was 17.7 percent. However, the late relapse rate was 18.5 percent and 7.8 percent, respectively. Van Slyke *et al*¹²⁷ used it in 64 previously untreated cases with 87.5 percent apparent cures and in 50 patients who had

¹²¹ Morse, Chas. E., and Hirsch, F. G.: Treatment of acute gonorrheal urethritis, *Mil. Surgeon*, **86**: 53, January 1940.

¹²² Committee for the Cooperative Clinical Survey of the Treatment of Gonorrhea: Gonorrhea in the Male: Results of Treatment with Sulfanilamide, General Scientific Meeting, Ninety-First Annual Session, A. M. A., June 11, 1940.

¹²³ Control of Venereal Disease in the War, London, December 16, 1939: Foreign Letters, J. A. M. A., **114**: 266, January 20, 1940.

¹²⁴ Westphal, L., Charles, R. L. and Carpenter, C. M.: Development of sulfapyridine-fast strains of the gonococcus, *J. Bact.*, **39**: 47, January 1940.

¹²⁷ Van Slyke, C. J.; Mahoney, J. F. and Wolcott, R. R.: Sulfapyridine treatment of gonococcal infection in hospitalized males: *Tr. Am. Neisserian M. Soc.*, **1939**: 120.

previously failed to respond to sulfanilamide, there were 76 percent apparent cures. They found concentrations of 4 to 6 mg. percent equally as effective as 11 to 14 mg. percent. In another group ¹²⁸ of 80 male patients with gonorrheal urethritis, treated with sulfapyridine, 79.2 percent passed all the usual tests for cure, and the average duration of discharge in the cured cases was 2.77 days. In a group of 19 patients previously resistant to another sulfonamide, 68.4 percent were apparently cured with sulfapyridine. These authors tabulated the experience of 10 other series, totaling 755 cases, in which the successful results ranged from 80 to 100 percent except in three relatively small series of cases in which successful results totaled 48, 62.5 and 70 percent, respectively. The average duration of discharge in four groups, of this series, varied from 3.3 days to 4.5 days.

The consensus of clinical opinion considers sulfapyridine the most efficient sulfanilamide derivative in the treatment of gonococcus infections. The one exception to this statement involves the use of sulfathiazole which was only recently released for general use. In a small series of cases Cook ¹³⁰ found that it resulted in clearing of the urethral discharge in all cases in 3 to 4 days at most and he considered it the drug of choice in these infections.

It is no longer considered necessary or advisable to begin sulfanilamide therapy with a small dosage and build up to a higher dosage in a few days. The maximum daily dose can be started when the diagnosis is made. Long continued medication appears equally useless and even harmful since it is these cases which develop granulocytopenia. Pelouze ¹²⁴ found no significant difference in results during the first 30 days if 400 grains of sulfanilamide were given in 1 week or over a period of 3 weeks. However, those cases receiving less than 400 grains of sulfanilamide during this period showed only one-sixth as many apparent cures and the results were no better than with local treatment alone. He concluded the drug might as well be discontinued if the patient has received no substantial improvement in 1 week and that after 3 weeks of sulfanilamide therapy there is no need to give it further. The cooperative group ¹²⁴ further found that 1 of 3 cases will classify as a relapse if cure is judged by the 2-glass test only; 1 of 20 will classify as a relapse if smear study is added and only 1 in 100 can be classified as a relapse when criteria for cure include the 2-glass test, smear and culture.

An adequate dosage of sulfapyridine is 45 grains daily. If no improvement occurs after 3 days this should be increased to 60 grains daily ¹²⁰. Blood levels of 2.8 mg. per 100 cc. have been found satisfactory ¹²⁸ and this was obtained with a dosage of 3 grams daily for 4

¹²⁸ Johnson, S. H. et al: Use of sulfapyridine in the treatment of gonococcal urethritis in the male, *Am. J. Med. Sc.*, **106**: 594-602, November 1939.

days, then 2 grams daily for 6 to 10 days. Very few workers continue the administration of this drug for more than 10 days for it is more toxic than sulfanilamide and tends to precipitate in the renal tubules. If no improvement has occurred after 1 week it is preferable to discontinue the drug. It may be repeated after 1 week's rest if desired.

Preliminary study ¹²⁰ indicates that sulfathiazole is highly effective in dosage of 60 grains daily for 10 to 14 days. It is considered superior ⁹¹ to either sulfanilamide or sulfapyridine in chronic, therapy-resistant cases. Before the advent of sulfanilamide it was frequently noted that certain patients failed to develop resistance to the gonococcus and their infection persisted for months. With sulfanilamide therapy a percentage of cases continue to be refractory to treatment. The development of drug-fastness in vitro ¹²⁹ suggests that the organism may acquire tolerance for the drug during treatment. This would explain the clinical course in those cases which become discharge free and after a few days, while still under treatment, again develop a profuse discharge. There are other cases which, insofar as can be determined, remain unaffected by drug therapy and it would seem that these strains of gonococci are drug resistant ¹³⁰. It has been frequently observed that a patient's infection may be resistant to one drug of this series and responsive to another. For this reason it is advisable in all therapy-resistant cases to give repeat-therapy with some other sulfonamide.

It is held ¹³¹ that sulfonamide therapy results in changes in the characteristics of the gonorrheal smear, the most striking change being the rapid decrease in the number of organisms, ¹³² which renders smear diagnosis difficult.

No change in morphology ¹³³ or biologic characteristics ¹²⁸ has been noted. In Service practice the smear diagnosis of recent cases is a simple matter. It is in the differential diagnosis of secretly and partially treated cases where smears prove inadequate. The cultural characteristics of the gonococcus are apparently not influenced by sulfonamide therapy. Cultures of prostatic exudates or secretions are more than 5 times as efficient as smears in determining the persistence of infection in asymptomatic cases. ¹³⁴

In gonorrheal ophthalmia these drugs have proven of great value. The incidence of corneal lesions is reduced. The average duration of

¹²⁰ Westphal, L., Charles, R. and Carpenter, C. M.: Development of sulfapyridine-fast strains of the gonococcus, *Ven. Dis. Info.*, 21: 183-187, June 1940.

¹²⁹ Felke: Reaction of gonococci to chemotherapy, *Klin. Wchnschr.*, 18: 568 April 22, 1939. Abst. in *Ven. Dis. Info.* 20: 216, July 1939.

¹³⁰ Jones, W. R.: *Am. J. Syph., Gonorr. & Ven. Dis.*, 22: 349, 1938.

¹³¹ Vest, S. A.; Hill, J. H., Harrill, H. C., Pitts, A. C.: Studies in the use of sulfanilamide in gonorrhea, *J. Urol.*, 40: 698, 1938.

¹³² Boak, R. A.; Charles, R. L. and Carpenter, C. M.: Tolerance of the gonococcus in vitro for increasing concentrations of sulfanilamide, *Tr. Am. Neisserian M. Soc.*, pg. 118, 1939.

¹³³ Cox, O. T., McDermott, M., and Hinton, W. A.: Evaluation of smear and cultural methods for detecting the gonococcus in males treated with sulfanilamide, *Tr. Am. Neisserian M. Soc.*, pg. 79, 1939.

the disease after starting treatment is reduced to 8.3 days as compared to 25.5 days prior to the use of sulfanilamide. It is said that once the smears become negative they remain so. This is compared to the finding of positive smears 28 days after cessation of discharge in non-sulfanilamide treated cases ¹³⁵

The treatment of gonococcal vaginitis in children has given no really striking results. In a recent study ¹³⁶ 68 percent of untreated cases were considered to undergo a spontaneous cure. The rest become carriers. During the carrier state, no clinical signs were present, smears were negative, and only some of many vaginal cultures taken were positive. Sulfanilamide produced a very rapid cure in about two-thirds of hospitalized patients. The responses occurred within 2 weeks after treatment was begun. Of 19 children given sulfapyridine all responded within 1 week. No follow-up studies had yet been made to determine the permanency of cure.

In the treatment of gonococcal arthritis Keefer ¹¹⁸ finds these drugs superior to older forms of therapy. The results are less striking in those cases with a local focus, sterile joint effusions, and low antibody response. Since complete recovery is dependent on rapid relief, before joint destruction occurs, Cook ¹²⁰ advocates fever therapy if no definite improvement has occurred after 48 hours. It is of interest to note the present practice in the British Army. ¹²⁵ In the 4 years of the World War 400,000 cases of venereal disease occurred in the British Army and the practice was to evacuate each case from his unit to a base hospital for venereal diseases. In the present war, except in cases presenting serious complications, all men in the early stages of syphilis, or suffering from acute anterior gonococcal urethritis, or soft sore, are treated in the field and not evacuated. At the base is a large hospital for venereal diseases where complicated cases, including those of untoward effects of therapy, are treated.

The routine treatment of gonorrhea is daily posterior irrigations with 1 : 8000 KMnO₄ and 3 grams daily of sulfapyridine for the first week and 2 grams daily for the second week. If at the end of the second week there is still some urethral discharge or the urine is not clear, the treatment is continued for another week. Testing for cure is carried out over a period of 10 weeks.

CHANCROID INFECTIONS

In this disease, caused by the hemophilic streptobacillus of Ducrey, sulfanilamide has proven very effective.¹⁰⁷ Further experience ^{127 138}

¹³⁵ Barbour, F. A., and Towsley, H. A.: Experience with sulfanilamide in treatment of gonorrheal ophthalmia, *Arch. Ophthalm.*, **22**, 581-589, October 1939.

¹³⁶ Cohn, A.; Steer, A.; and Adler, E. L.: Gonococcal vaginitis, *Ven. Dis. Info.*, **21**, 208-220, July 1940.

¹³⁷ Schwartz, W. F., and Freeman, H. E.: Sulfanilamide in the treatment of chancroid, *J. A. M. A.*, **114**: 946, March 16, 1940.

¹³⁸ Greenblatt, R. B., and Sanderson, E. S.: Sulfanilamide in chancroid diseases, *Am. J. Syph., Gonorrhea & Ven. Dis.*, **23**: 605-612, September 1939.

confirms the earlier observations. In moderate dosage the ulcers and bubo respond in approximately 2 weeks.

LYMPHOGRANULOMA VENEREUM

This widespread disease, clinically mild in its early stages, is productive of chronic invalidism with the development of rectal stricture, esthiomene and granulomatous lesions in the colon. With no effective remedy available it is particularly gratifying to note the recent encouraging reports with the use of various sulfanilamide derivatives. The tests with sulfathiazole and sulfapyridine in a large number of mice against the agent of lymphogranuloma venereum show both to have an appreciable therapeutic effect.⁴ The results agree with those obtained by other investigators with sulfanilamide. In one series¹³⁹ of 14 cases, diagnosed by positive Frei tests, rectal stricture and high globulinemia, either sodium sulfanilyl sulfanilate ($\text{NH}_2\text{C}_6\text{H}_4\text{SO}_2\text{NH}\text{C}_6\text{H}_4\text{SO}_2\text{Na}$) or sodium sulfanilate ($\text{NH}_2\text{C}_6\text{H}_4\text{SO}_2\text{Na}$) were given intravenously for 6 to 35 weeks. There was cessation of the bloody purulent discharge, disappearance of fistulas and absorption of rectal stricture. In 4 cases previous colostomy openings healed and normal bowel function was restored. With most of the cases under observation for over a year the Frei test remained positive but the rectal stricture and general health showed improvement. Other cases were treated by oral administration which appeared equally effective. A blood level of 2.5 mg. percent of sodium sulfanilate was adequate.

In another series¹⁴⁰ using sulfanilamide, it was noted that the patients' general health improved noticeably, the scar tissue became more elastic, the perirectal infiltration and exudate disappeared, raw surfaces healed and resistance to the examining finger diminished. Used in the acute cases¹⁴¹ a prompt response has been noted with progressive decrease in pain and swelling of the involved glands and closure of the draining areas. Curth and Sanders¹⁴² report a case of conjunctivitis due to this virus. The lesion was rendered inactive through the use of sulfanilamide over a period of 4 months.

NONVENEREAL UROLOGIC DISEASES

In the simple uncomplicated cases of infection of the urinary tract any of the sulfanilamide derivatives will effect cure in 90 percent of cases.¹²⁰ The average case does not warrant thorough cystoscopic and laboratory study and small or moderate dosage is adequate. However, if symptoms persist after one or two courses of treatment,

¹³⁹ Hebb, A.; Sullivan, S. G., and Felton, L. D.: Treatment of lymphopathia venereum with sodium sulfanilyl sulfanilate and sodium sulfanilate. Pub. Health Rep., 54: 1750, September 29, 1939.

¹⁴⁰ Marino, A. W. M.; Buda, A. M.; Turell, R., and Nerb, L.: Use of sulfanilamide for lymphogranuloma inguinale, Amer. J. of Surg., 46: 343, November 1939.

¹⁴¹ Trautman, J. A., and Thomason, H. A.: New Orleans Med. and Surg. J., 92: 441, February 1940.

¹⁴² Curth, Wm.; Curth, H. O., and Sanders, M.: Chronic conjunctivitis, J. A. M. A., 115: 445-447, August 10, 1940.

bacteriologic and pathologic studies become necessary. The usual causes of failure are lesions as stricture, stones, tumor, prostatic hypertrophy, or diverticulum, any of which require surgical intervention, and mixed or drug-resistant infections and pregnancy.

In the usual case 40 grains of sulfanilamide daily for 8 to 10 days will effect cure. This drug is preferred to sulfapyridine for it is equally effective and less toxic.⁷ If necessary, after a 2-week rest period, the drug can be repeated. Sulfathiazole is said to be the more valuable drug in chronic pyelitis cases.¹¹⁸ It destroys the usual gram negative organisms in addition to the staphylococcus and *S. fecalis* which are resistant to sulfanilamide and sulfapyridine. It is also effective against the pseudomonas organisms.^{7 42}

In treating *S. fecalis* infections a urine pH of 5 or below is desirable, for the acidity is important, as shown experimentally (Helmholtz) and clinically (Cook). Against other organisms the bactericidal activity of urine containing sulfanilamide increases when the hydrogen-ion concentration is changed from pH 6.2 to 7.7.¹⁴³ There is also a great variation in the level at which sulfanilamide acts bactericidally on different strains of the same groups of organisms commonly found in the urinary tract. Alyea¹⁴⁴ believes that failures with a drug are due to strain resistance and if a case does not react favorably in 3 days another drug should be tried. He reports the same percentage of cures whether fluids are restricted to 1,800 cc. daily or allowed up to 4,000 cc. This should encourage the liberal use of fluids, especially with sulfapyridine and sulfathiazole,¹¹⁹ and thereby lessen the danger of drug precipitation in the urinary tract.

TOXIC REACTIONS

The various toxic manifestations which occur with the use of these drugs have been adequately presented in the medical literature. A recent review,⁶³ worthy of reading in its entirety, presents the percentage incidence of toxic reactions and the time during therapy when each may be expected. Many of the reactions induce only slight malaise, are of minor importance and require only reassurance. It is preferable to discontinue therapy with the appearance of drug fever, dermatitis, hematuria, granulopenia and neuritis. The more severe reactions requiring cessation of therapy include icterus, hepatitis, acute hemolytic anemia, anuria, gross hematuria, purpura, psychosis and impending agranulocytosis all of which are relatively rare. With the exception of acute leukopenia the toxic manifestations occurring in the first 2 weeks of therapy can be recognized by careful

¹⁴³ Sickler, J. R.: Effect of pH on the bactericidal power of urine containing sulfanilamide, *Proc. Staff Meet. Mayo Clinic*, 14: 715, November 8, 1939.

¹⁴⁴ Alyea, E. P.: Present status of chemotherapy in nonspecific infections of the urinary tract, *Scientific Session, Section on Urology, A. M. A.*, June 12, 1940.

clinical observation.⁶³ The malignant form of granulocytopenia occurs rarely, yet it is the cause of a large proportion of the deaths from this therapy. It seldom occurs with less than 2 weeks of treatment and therefore need be of little concern in most cases which will require treatment for 10 days or less. If treatment is continued for a longer period a white blood count should be obtained on alternate days.

The renal complications of sulfanilamide are rare. With sulfapyridine¹⁴⁵ and sulfathiazole¹⁴⁶ therapy, hematuria and anuria are clinical danger signals and an accurate measurement of the urine passed in each 24 hours is therefore of importance.^{63 147} The renal complications follow crystalline precipitation of these relatively insoluble drugs, and of their even more insoluble acetylated derivatives.^{148 149} These crystals, even in large amounts, are not opaque to x-rays.^{150 151} They occur chiefly in the tubules where their mechanical effect causes hematuria.¹⁵¹ In itself, this is not of grave consequence since the changes are reversible. Not all hematuria occurring in acute infections is due to these drug concretions since 7 percent of 381 pneumonia patients³⁰ showed hematuria before sulfapyridine therapy, whereas in only 5.4 percent of 277 cases was hematuria discovered after starting treatment. On the other hand 40 percent of a series of pneumococcic pneumonia cases on sulfapyridine therapy were found to have a transient hematuria.¹⁵¹ The incidence of hematuria in acute upper respiratory infections was recently discussed in this Bulletin.¹⁵² However, a diffuse toxic nephritis may develop^{153 154} incident to sulfapyridine therapy. At times the concretions completely obstruct the urinary tract.¹⁵⁰ This can be relieved by ureteral catheterization and lavage with physiologic saline.^{63 150} In all such cases fluids should be forced. The forcing of fluids during sulfonamide therapy will tend to prevent toxic manifestations, especially those related to the kidneys. There are several reports indicating this practice does not affect the maintenance of adequate therapeutic levels of the drug.

¹⁴⁵ Plummer, H. and McLellon, M.: *J. A. M. A.*, **114**: 943, 1940. Stryker, W. A.: *J. A. M. A.*, **114**: 953, 1940.

¹⁴⁶ Pepper, D. S. and Horack, H. M.: *Am. J. Med. Sc.* **199**: 674-680, May 1940.

¹⁴⁷ Brown, W. H.; Thornton, W. B. and Wilson, J. S.: *J. Clin. Invest.*, **18**: 803 (Nov.) 1939.

¹⁴⁸ Antopol, Wm., and Robinson, H.: *Proc. Soc. Exper. Biol. & Med.*, **40**: 428, March 1939.

¹⁴⁹ Gross, Paul; Cooper, F. B., and Lewis, M.: Fate of urinary calculi caused by the administration of sulfapyridine, *Urol. & Cutan. Rev.*, **43**: 439, July 1939.

¹⁵⁰ Carroll, G.; Shea, John and Pike, Geo.: Complete anuria due to crystalline concretions following the use of sulfapyridine in pneumonia, *J. A. M. A.*, **114**: 411-412, February 3, 1940.

¹⁵¹ Antopol, W.: Occurrence of urologic complications in humans following sulfapyridine therapy, *J. Urol.* **43**: 589-598 April 1940.

¹⁵² Funk, Wm. H.: Hematuria, associated with acute upper respiratory infection, *U. S. Nav. Med. Bull.* **38**: 311, July 1940.

¹⁵³ Kilgour, J. M.: *Canad. Med. Assoc. J.*, **41**: 445, November 1939.

¹⁵⁴ Smith, F. J., and Needles, R. J.: Report of 50 cases of acute lobar pneumonia in adults treated with sulfapyridine, *Am. J. Med. Sc.*, **193**: 19-22, July 1939.

These drugs diffuse throughout all body fluids, including those of the fetus, in approximately equal concentrations. Prolonged administration to pregnant rats causes increased intra-uterine and postnatal mortality, decreased litter size, diminished birth weight, and selective stunting of growth.¹⁵⁵ The clinical application of this experimental work was commented upon by Pratt and Stewart.¹⁵⁶ Apparently the usual clinical dosage is not injurious in the human. At least no harmful effects from sulfanilamide have been reported in the human fetus.¹⁵⁷ It is well to remember the drug should not be given during pregnancy for relatively harmless conditions.

The cyanosis in man after sulfanilamide therapy is explained by the presence of methemoglobin (rarely sulfhemoglobin).^{158 159} Neither cyanosis nor methemoglobin has been noted following either sulfapyridine¹⁵⁸ or sulfathiazole medication.⁶³ The administration of methylene blue will abolish cyanosis due to methemoglobin. It can be given orally in doses of 1 grain every 4 hours in children weighing 20 kilograms or less and up to 2 grains in adults. This drug is an irritant to the gastro-intestinal tract and is not necessary in most cases.

The occurrence of porphyrinuria incident to sulfonamide therapy and its relation to the toxic manifestations has occasioned some comment. Rimington¹⁶⁰ observed that patients given sulfanilamide show an increased elimination of coproporphyrin in the urine and that acute porphyrinuria is associated with nausea, vomiting, colic, constipation, and muscular weakness. He suggested that it is the variable proportion of the photosensitizing coproporphyrin which may account for dermatitis in some patients. It had been noted,¹⁶¹ though this is now refuted as a constant finding,¹⁵ that pellagrins have porphyrinuria. This was followed by the demonstration¹⁶² (also refuted)¹⁶³ that the porphyrinuria of pellagrins returned to normal incident to nicotinic acid therapy. The same was found true of the porphyrinuria associated with radiation sickness in which the nicotinic acid promptly alleviated nausea, vomiting, anorexia, and headache. The similarity of these symptoms to those commonly

¹⁵⁵ Speert, H.: Placental transmission of sulfanilamide and its effects on the fetus and newborn, *Bull. Johns Hopkins Hosp.*, **66**: 139, March 1940.

¹⁵⁶ Pratt, J. P., and Stewart, H. L., Jr.: Is sulfanilamide dangerous to babies? *J. A. M. A.*, **114**: 1687, April 27, 1940.

¹⁵⁷ Adair, Fred L.: Use and abuse of chemotherapy in obstetrics and gynecology, symposium on chemotherapy, Scientific Session, *A. M. A.*, June 11, 1940.

¹⁵⁸ Vigness, Irwin; Watson, C. J.; and Spink, W. W.: Relation of methemoglobin to the cyanosis observed after sulfanilamide administration, *J. Clin. Invest.*, **19**: 83, January 1940.

¹⁵⁹ Hartman, A. F., Perley, A. M., and Barnett, H. L., *J. Clin. Invest.*, **17**: 699, November 1938.

¹⁶⁰ Rimington, C.: Porphyrinuria following sulfanilamide, sulfanilamide dermatitis, *Lancet*, **1**: 770, April 2, 1938.

¹⁶¹ Spies, T. D.; Cooper, C.; and Blankenhorn, M. A.: Use of nicotinic acid in treatment of pellagra, *J. A. M. A.*, **110**: 622, February 26, 1938.

¹⁶² Spies, T. D.; Bean, Wm. B.; and Stone, R. E.: *J. A. M. A.*, **111**: 584, August 13, 1938.

¹⁶³ Rosenblum, L. A., and Jolliffe, N.: Porphyrinuria in pellagra, *Am. J. Med. Sc.*, **199**: 853-858, June 1940.

associated with sulfanilamide therapy was recognized ¹⁶⁴ and nicotinic acid therapy instituted. The most marked change in these sulfanilamide-treated cases was a clearing of their mental apathy, a decrease in the usual unpleasant symptoms, and in the porphyrinuria. There have been other reports of encouraging results from the use of nicotinic acid in relief of these toxic reactions ^{30 165} and in combating the vomiting incident to sulfapyridine therapy.¹⁶⁶

These drugs tend to produce giddiness, faintness, difficulty in concentration and disorientation which interfere with the operation of automobiles and airplanes,^{167, 168}. A full dose taken shortly before flying is said to lower an aviator's ceiling by about 5,000 feet.¹⁶⁹ For this reason passengers and aircraft employees, while under such therapy are prohibited from flying in England.¹⁷⁰ At least one airplane accident in this country and possibly several others have been attributed to this cause.¹⁷¹

Sulfanilamide has recently been advocated ¹⁷² for use as a bacteriostatic agent in stored blood. In this connection attention should be called to a report ¹⁷³ of two cases in which sulfanilamide treatment was followed by an alteration in the blood serum which precluded the finding of a suitable donor for blood transfusion. Following this experience normal serum was treated in vitro with sulfanilamide. After 4 days this serum agglutinated the red blood cells of donors of the corresponding blood group. Long ⁶³ reports 2 deaths within 15 minutes following the injection of sodium sulfapyridine through the rubber tube of a transfusion apparatus. Since this experience, he advocates the drug not to be added to the blood during transfusion. This should not be interpreted, however, as contraindicating transfusion of a patient receiving sulfonamide therapy.

It is customary in using sulfapyridine to administer bicarbonate of soda, gram for gram, with the idea of rendering urine so alkaline that

¹⁶⁴ McGinty, Park; Lewis, G. T., and Holclaw, M. R.: Symptoms occurring with sulfanilamide relieved by nicotinic acid, *J. M. A. Georgia*, **28**: 54, February 1939.

¹⁶⁵ Doughty, J. F.: Sulfanilamide cyanosis relieved by nicotinic acid, *J. A. M. A.*, **114**: 756, March 2, 1940.

¹⁶⁶ Brown, W. H.; Thornton, W. B., and Wilson, J. S.: Clinical toxicity of sulfanilamide and sulfapyridine, *J. A. M. A.*, **114**: 1605-1611, April 27, 1940.

¹⁶⁷ Long, P. H.: Clinical use of sulfanilamide and its derivatives with special reference to their possible toxic effects, *Ohio State Med. Jr.*, **34**: 977-981, 1938.

¹⁶⁸ McGinty, A. P.: Review of the complications following the administration of sulfanilamide, *Jr. Med. Assoc. Georgia*, **27**: 21-29, 1938.

¹⁶⁹ Mackie, F. P.: Sulfanilamide in the air: A warning, *Brit. M. J.*, **2**: 139, July 15, 1939.

¹⁷⁰ Foreign Letters: Effect of sulfonamide derivatives on aircraft pilots, *J. A. M. A.*, **113**: 1237, September 23, 1939.

¹⁷¹ Correspondence: Effect of sulfonamide derivatives on airplane pilots, *J. A. M. A.*, **114**: 1478, April 13, 1940.

¹⁷² Novak, M.: Preservation of stored blood with sulfanilamide, *J. A. M. A.* **113**: 2227-2229, December 16, 1939.

¹⁷³ Scott, B. A., and Meerapfel, O.: Effect of sulphonamides on blood serum, *Lancet*, **2**: 244-246, July 29, 1939.

the precipitation of acetylsulfapyridine would be hindered. Bensley¹⁷⁴ has studied the solubility of both free and conjugated sulfapyridine and considered changes in solubility, as alkalinity increased, to be too small to make alkalization effective in preventing precipitation of the drug from the urine. Alkalies continue to be given, however, and their value or necessity remains to be determined.

The introduction of sulfathiazole has brought out two new types of reaction.¹⁷⁵ One is an erythema nodosum type of skin eruption and the other a conjunctival and scleral injection.

SUMMARY

There has been no attempt to cover the controversial and theoretical question of mode of action of these drugs. In the present state of our knowledge this need not concern the clinician.

In presenting this review an attempt has been made to stress the clinical problems. The subject matter is an extension of remarks previously appearing in this publication. The four compounds used most widely at this time have been considered in some detail. Their application to the therapy of infectious diseases has been reviewed and the experience of various workers cited. For those interested in more detailed information the references may prove a guide to further reading.

SULFAPYRIDINE TREATMENT OF PNEUMONIA

EMPLOYED ALONE AND COMBINED WITH SERUM

By Lieutenant (Jr. Gr.) R. O. Canada, Medical Corps, United States Navy

The purpose of this article is to report on 50 cases of lobar pneumonia treated with sulfapyridine or serum and sulfapyridine at the Norfolk Naval Hospital during the winter of 1939-40. Included in these are 43 enlisted men, 1 officer, 3 women, and 3 children. The oldest case was 36 years and the youngest was 2 years of age. The average age was 21.2 years.

The length of service of naval personnel is given in table 1. As shown by this table 44 percent had less than 4 months service and 60 percent had less than 6 months service. The majority of these men were from Southern States and were unaccustomed to the prevailing climatic conditions which may account for the fact that more recruits developed pneumonia than men with longer service. The cases were admitted early in the disease, on an average of 2.4 days after the onset of illness. In view of this and the fact that all cases were under 36 years of age, a low mortality would be expected.

¹⁷⁴ Bensley, E. H.: Toxic effects of sulfanilamide and related compounds, Canadian Med. Assoc. J., **42**: 30, January 1940.

¹⁷⁵ Haviland, J. W. and Long, P. H.: Skin, conjunctival and scleral reactions in course of therapy with sulfathiazole, Johns Hopkins Hosp. Bull., **66**: 313-319, May 1940.

TABLE 1.—*Length of service of naval personnel*

Service	Number	Percent	Service	Number	Percent
0 to 2 months.....	5	12	10 to 12 months.....	1	2
2 to 4 months.....	14	32	Over 1 year.....	12	27
4 to 6 months.....	7	16	Total.....	44	100
6 to 8 months.....	3	7			
8 to 10 months.....	2	4			

A pneumococcus type was found in the sputum of 33 cases and there were 13 different types in all. These are shown in table 2. All cases reported in this series had x-ray evidence of lobar pneumonia at time of admission.

TABLE 2.—*Pneumococcus types and complications*

Type.....	I	III	IV	V	VII	VIII	X	XV	XVI	XVII	XVIII	XXI	XXXII	(e)	Total
Number.....	6	4	1	2	8	3	2	1	1	1	2	1	1	17	50
Complications..	1	0	1	1	2	2	1	0	0	0	0	0	0	5	13

• No type established.

PROCEDURE

It was the rule that as soon as a case of suspected pneumonia was admitted the following procedures were carried out:

1. Physical examination.
2. X-ray of chest.
3. Complete blood count.
4. Pneumococcus typing.
5. Blood culture.
6. Urinalysis.

Neither serum nor sulfapyridine was given until reports from these examinations had been completed. Antipneumococcus serum was kept on hand in lots of 200,000 units for each of the usual pneumococcus types seen in this locality. A standing order was left with the Officer-of-the-Day to send by dispatch to the supply depot for an additional 200,000 units of serum when a case of pneumonia was admitted and the type established. By doing this a sufficient supply of sera for the usual types was kept on hand and serum for the unusual type could be obtained within 24 to 36 hours.

Serum and sulfapyridine were administered to all cases in which a pneumococcus type could be established except in six cases in which the type was not recovered from the sputum until after the patient had begun to improve under sulfapyridine therapy.

Oxygen tents were used with all except the very mild cases. It was the policy to administer oxygen early in the course of the disease rather than to wait until definite indications arose. Oxygen was of particular value in relieving the following conditions: (a) Anoxemia, (b) pleural pain, and (c) nausea (from sulfapyridine). Also, tents provided a cool atmosphere for which the feverish patient was very grateful.

SULFAPYRIDINE

The routine with adults was to give 2 grams of sulfapyridine as the initial dose, followed in 4 hours by 2 grams, and 1 gram every 4 hours thereafter, until the temperature had been normal for a period of 72 hours. The children were given doses calculated on the basis of 1 grain per pound body weight per 24 hours. One half of the calculated amount of drug for 24 hours was administered as the initial dose.

The routine was not followed strictly in every case. Extreme toxicity, extensive consolidation, or a blood concentration below 3 mg./100 cc. was considered indication for increasing the amount usually given. The average total dose was 30.3 grams and the average length of time the drug was administered was 5 days. This dosage produced blood concentrations of free sulfapyridine which ranged from a trace to 18.5 mg./100 cc., the average being 6.95 mg./100 cc. There was no strict relationship between the dosage and concentration of free sulfapyridine in the blood. In several cases large doses failed to raise the blood concentration to the high levels observed in patients who received smaller amounts of the drug.

Some degree of nausea was experienced by 90 percent of the cases. In only one instance was the nausea so severe that the drug had to be discontinued and then only for a period of 24 hours. The various methods used to combat nausea were bicarbonate of soda, tincture of belladonna, phenobarbital, oxygen, intravenous dextrose, and aluminum hydroxide liquid (Creamalin). The efficiency of these methods were as in the order given, soda being least effective and aluminum hydroxide liquid most effective. Aluminum hydroxide liquid was administered in 1-ounce doses 30 minutes before each sulfapyridine medication and sulfapyridine tablets were ground and given in milk. This method proved effective in relieving nausea when all else had failed.

The toxic reactions to sulfapyridine were hematuria and flank pain in two cases, hemolytic anemia in one case, and agranulocytosis in one case. One death was considered caused by the precipitation of acetyl-sulfapyridine crystals in the urinary tract with subsequent urinary retention and uremia. This case is presented below.

SERUM

The intradermal and conjunctival tests were used in testing for sensitivity to serum. If no sensitivity was demonstrated serum was administered 2 cc. (approximately 20,000 units) as the initial dose and 4 cc. at 4-hour intervals until a total of 10 cc. or approximately 100,000 units had been given. If the temperature was not normal or tending toward normal at the end of 24 hours, a second 10 cc. dose was given at one time. Three cases presented immediate allergic

reactions to serum, one with chills, vomiting, and temperature elevation to 105.6, and two cases had immediate urticaria. Three cases presented delayed reactions. The symptoms were almost identical in the three cases, i.e. fever, joint pain, generalized aching, and lymphadenopathy which lasted 4 days in each case.

RESULTS

An unselected group of 50 cases was treated with sulfapyridine or serum and sulfapyridine during the winter 1939-40. All except 3 cases were admitted before the fourth day of illness. A drop in temperature and decrease in toxicity occurred on the average of 31.7 hours after treatment was instituted. The leucocyte count, pulse, and respiratory rates almost paralleled the temperature curves. It was amazing to see a patient acutely ill and toxic running a high fever one day and the following day find the same patient with normal temperature, without signs of acute infection, and asking if he might sit up in bed. This often occurred within 24 hours after admission. When there were definite physical signs of consolidation at time of admission, although the temperature returned to normal and signs of toxicity disappeared shortly after starting treatment, the chest did not begin to clear until an average of 4 days had elapsed from the first sign of improvement. For this reason it was thought best to continue treatment with sulfapyridine for at least 3 days after the temperature had returned to normal. There were 2 cases in which sulfapyridine was discontinued because of drug toxicity when the first sign of improvement had appeared. These 2 cases had a subsequent spread of the infection. There were 10 cases with x-ray evidence of pneumonia on admission and without definite physical signs of consolidation. These 10 cases never developed the typical physical signs of pneumonia.

COMPLICATIONS

In this series there were 13 complications to pneumonia. Five developed otitis media, 5 pleural effusion, 2 chronic fibrinous pleurisy, and 1 empyema. The empyema case ran a high sustained temperature from the outset in spite of large doses of sulfapyridine with an average blood concentration of 13.6 mg./100 cc. No pneumococci could be found in the sputum. Signs of fluid in the chest were first noted on the tenth hospital day. Fluid aspirated from the chest revealed a pure culture of *Staphylococcus aureus*. Surgical drainage was established and the patient made a slow recovery.

Only one case had a positive blood culture. Serum and sulfapyridine were given and the temperature returned to normal 14 hours after treatment was begun. A blood culture taken at the end of 24 hours was negative.

SULFAPYRIDINE *vs.* SERUM AND SULFAPYRIDINE

As shown by table 3 those cases receiving only sulfapyridine responded to treatment as quickly as those receiving both serum and sulfapyridine. Fifty-two percent of the cases in each group had a normal temperature within 24 hours after treatment was begun. There was no relation between the blood concentration of free sulfapyridine and the clinical response. Complete recovery and return to duty was accomplished on an average of 6.5 days sooner in the sulfapyridine group than in the group which received both serum and sulfapyridine. There were eight complications in the serum and sulfapyridine group as compared to five in the sulfapyridine group. It should be remembered that this series of cases is too small to draw conclusions from these observations.

TABLE 3.—*Serum and sulfapyridine vs. sulfapyridine*

Interval in hours until temperature became normal	Serum and sulfapyridine			Sulfapyridine		
	Cases		Average sulfapyridine blood concentration mg./100 cc.	Cases		Average sulfapyridine blood concentration mg./100 cc.
	Number	Percent		Number	Percent	
0 to 12.....	2	7	10.1	5	22	7.7
13 to 24.....	12	45	6.3	7	30	8.4
25 to 36.....	3	11	7.6	3	13	6.7
37 to 48.....	4	15	7.6	5	22	8.6
49 to 60.....	2	7	11.2	0	0	-----
Over 60.....	4	15	5.0	3	13	5.4
Total.....	27	-----	-----	23	-----	-----

SPECIFIC TREATMENT COMPARED WITH NONSPECIFIC TREATMENT

Seventy cases of lobar pneumonia which received no specific treatment were taken from the files of the Norfolk Naval Hospital for the years 1935-37, inclusive. The average age was 22.5 years and only two cases were over 40 years of age. Out of this group there were seven deaths, a mortality of 10 percent. An average of 63.6 days per patient were lost from duty. Of the present 44 cases of naval personnel which received either serum, sulfapyridine, or both the average age was 21.5 years and all cases were under 40 years of age. There was one death. An average of 25.2 days per patient were lost from active duty.

CASE REPORTS SHOWING DRUG REACTIONS

Case 1. (J. R.), a man aged 25 was admitted to Norfolk Naval Hospital on the second day of illness. Physical and x-ray examinations showed consolidation of the right middle lobe. A type III pneumococcus was recovered from the sputum. Blood cultures were negative. Complete blood count on admission was reported: RBC. 4,200,000, Hgb. 90%, WBC. 14,800, juveniles 12%, bands 38%, segmented 42%, lymphs. 7%, and monos. 1%.

The patient was irrational at times and appeared quite toxic. Treatment was started immediately and 150,000 units of type III antipneumococcus serum were administered within the first 15 hours after admission. Sulfapyridine was given, 10 grams per day for the first 2 days and 6 grams per day thereafter. The temperature returned to normal 5 days after treatment was begun. On the 7th hospital day the patient began to run a low-grade fever. A spread of the infection to the right lower lobe was found. On the 17th hospital day and after a total of 104 grams of sulfapyridine had been administered, a blood smear revealed only 22% granulocytes. All medication by mouth was discontinued. Four days later the total white count had dropped to 2,000 with only 5% granulocytes. The red blood cell count was 2,700,000, hemoglobin 60%, and platelet count 307,000. The patient complained of sore throat and sore gums. On examination the throat was quite inflamed and edematous. The anterior cervical glands were enlarged and tender to palpation. The gums had a white spongy appearance with a red line at the dental margin. For the following 3 days granulocytes completely disappeared from the blood smear. Blood transfusions and intramuscular injections of 10 cc. pentnucleotide were given daily. On the 25th hospital day early granulocyte forms began to appear in the blood smears. The granulocytes and total white count increased daily and by the 31st hospital day the blood picture was reported: RBC. 4,600,000, Hgb. 87%, WBC. 13,000, bands 19%, segmented 35%, lymphs. 28%, and monos. 18%. The chest cleared rapidly and the patient had no further complications.

Case 2. (P. H., Jr.), a man aged 18 was admitted to Norfolk Naval Hospital on the second day of illness. Physical and x-ray examinations showed a lobar pneumonia involving the left upper lobe. A type X pneumococcus was recovered from the sputum and confirmed by the mouse inoculation test. During the first 4 days after admission 200,000 units of type X antipneumococcus serum were administered. Sulfapyridine was not retained when given by mouth and an attempt was made to give the soluble monohydrate form intravenously. Violent nausea and dizziness were experienced after the injection of only 0.6 gram and so this method was abandoned. Finally the patient was able to retain sulfapyridine given by mouth in milk preceded by aluminum hydroxide liquid. On the fourth hospital day a blood concentration of 6.8 mg./100 cc. was obtained. The temperature returned to normal 8 days after admission and sulfapyridine was discontinued; a total of 36 grams had been given. On the fourteenth hospital day the temperature rose to 102. The patient complained of pain in joints and itching skin. The face was flushed and puffy and there was a generalized glandular enlargement. In view of these symptoms and the fact that 12 days had elapsed since serum was first given, this was considered an allergic reaction to serum. However an x-ray taken of the chest showed a spread of the pneumococcal process to the left lower lobe. Sulfapyridine was started again but there was no improvement and the temperature remained elevated. On the eighteenth hospital day the urine showed 100 mg./100 cc. albumin and an occasional erythrocyte. Sulfapyridine was discontinued immediately, a total of 48 grams in all having been given. Gross hematuria appeared the following day. The blood NPN. was 41 mg./100 cc. and the urea nitrogen was 19 mg./100 cc. The urinary output decreased and the nitrogenous products of the blood increased. On the 22d hospital day a blood NPN. was reported 225 mg./100 cc. and urea nitrogen 100 mg./100 cc. Infusions of dextrose and saline were given. The patient was cystoscoped and catheters passed to both kidney pelves but only a small amount of bloody urine flowed from one catheter. The kidney pelves were irrigated with normal saline solution but there was no increase in the urinary flow. The patient lapsed into coma and died in uremia on the 23d day after admission. An autopsy was not permitted.

SUMMARY

1. In this article are presented 50 cases of lobar pneumonia treated in the Norfolk Naval Hospital with sulfapyridine or serum and sulfapyridine. There was only one death and this was thought caused by the drug used in treatment rather than by the disease. The mortality for the years 1935, 1936, and 1937 for approximately the same age group but which cases received no specific treatment, was 10 percent. All except 3 cases in this series were treated before the fourth day of illness. The complications to pneumonia were otitis media in 5, pleural effusion in 5, chronic fibrinous pleurisy in 2, and empyema in 1.

2. There was no strict relationship between the amount of sulfapyridine administered and the blood concentration. Nausea of some degree occurred in 90 percent of the cases. In only 1 case was this so severe that the drug had to be discontinued. The other manifestations of drug toxicity were hematuria, hemolytic anemia, and agranulocytosis. The 1 death in this series was thought caused by the precipitation of acetyl-sulfapyridine crystals in the urinary tract.

3. Of the 33 serum treated cases, 3 presented immediate allergic reactions and 3 had delayed reactions to serum, none of which was severe.

4. Temperature returned to normal and signs of toxicity disappeared on an average of 31.7 hours after treatment was begun. In cases that showed definite physical signs of consolidation, the chest did not begin to clear until an average of 4 days had elapsed from the first sign of improvement. This was taken as reason for continuing sulfapyridine medication for at least 3 days after the temperature had returned to normal.

5. In this series there was no advantage in using serum and sulfapyridine as compared to sulfapyridine alone.

6. An average of 25.2 days per patient were lost from active duty as compared to 63.6 days per patient in a group of 70 cases admitted in previous years which received no specific treatment.

SULFANILAMIDE THERAPYREPORT OF A NEW TYPE OF REACTION¹

By Lieutenant Commander Ray W. Hege, Medical Corps, United States Navy

The writer has been unable to find reports or references to reactions to sulfanilamide therapy involving the mucous membranes, except as would be found in cases of granulocytopenia resulting from use of the drug. The following case report is of a reaction to sulfanilamide

¹ From the U. S. S. *Concord*. Received for publication June 16, 1939.

therapy in which the mucous membrane of the mouth and penis was involved with possible involvement of the conjunctiva of the right eye near its junction with the true skin. At the time of the original examination of this patient on the first admission, granulocytopenia was thought to be a very likely diagnosis, but a blood examination and early recovery proved the condition to be very much less serious, and it was thought that the affection was a local infection. On the return of the symptoms after resumption of therapy, 2 months after the first treatment and symptoms, the author became convinced that they were the result of the treatment by sulfanilamide.

CASE REPORT

W. E. B., colored, age 27 was admitted to the sick list on May 3, 1939, with complaint of soreness of mouth, itching of right eye and itching of penis.

PRESENT ILLNESS.—The onset of complaint followed the use of sulfanilamide for 2 days and after having taken 140 grains of that drug, 20 grains 4 times a day. The first symptoms included soreness of the lips and tip of the tongue, itching of the right eye and of the penis. The soreness of the tongue was especially marked about the tip and at the tongue margins. The gums were also tender. The itching of the penis was intense and the patient stated that he thought the irritated lesions on his penis were due to his rubbing to alleviate the itching. A similar itching was complained of about his upper right eyelid. The soreness of the mouth became worse and eating became painful. No systemic symptoms accompanied these complaints as the patient felt normal otherwise.

PAST HISTORY.—The medical abstract shows *G. C.* infection of the urethra, February 23, 1938, from which the patient states that recovery was prolonged and that he had occasional discharge for many following months. During February 1939 he was given sulfanilamide for a urethral discharge which failed to show the gonorrhoeal organisms. At that time, after 4 days treatment, he was admitted to the sick list with glossitis. The symptoms were identical to those of the present admission except that they were more severe. At that time the tongue developed three large blisters which ruptured and remained as loose membranes until the entire dorsal surface of the tongue exfoliated, leaving a clear tender, healing surface which gradually returned to normal after the drug was discontinued. A similar itching and swelling was noted about the penis. After the mouth had completely healed there remained several lesions on the penis suggesting chancroidal infection. There was no adenopathy. Dark-field examinations made from the lesions of mouth and penis were negative for both Vincent's organisms and *Spirochaeta pallida*. At onset of glossitis, WBC., 9,100; polys. 60%; lymphs. 33%. All signs of acuteness subsided after 4 days under symptomatic treatment and discontinuation of the drug.

PHYSICAL EXAMINATION. *General.*—Tall, slender, male Negro, weighing about 170 lbs. Does not appear acutely ill. Temperature normal. Pulse normal. The examination was essentially negative except for the middle of the right upper eyelid which shows a small yellow crust about 4 mm. square at its lower margin. There is no appreciable ulceration. This lesion does not involve the under conjunctival surface.

Mouth.—The tongue presents a thick grayish coat over the dorsal surface which thins toward the margins where the tongue is red, slightly edematous and appears inflamed. The gums show a grayish film except near their junction with

the teeth where there is again redness and tenderness apparent. The buccal surfaces also show this grayish coat but there are irregular linear areas which show no coating, redness, or signs of inflammation. Neither pharynx nor tonsils appear to be involved.

Penis.—The penis shows some edema of the prepuce. There are three lesions present, about $\frac{1}{2}$ cm. in size on the ventral aspect near the frenum. One lesion is on the cutaneous surface, one over the margin involving both surfaces and the third involving the mucous surface only. Early they appeared as edematous weeping and slightly traumatized lesions and without definite borders. Later they developed into superficial ulcers with definite borders covered with yellowish crust. These ulcers healed readily without secondary infection. The remaining mucous surface of the prepuce appears edematous and a few superficial linear fissures developed which exuded the serous discharge. All these lesions of the penis apparently itched intensely. There was no adenopathy.

LABORATORY FINDINGS.—On admission and after 2 days of sulfanilamide therapy: WBC. 7,050; polys. 72%; lymphs. 24%; eosins. 1%; monos. 3%. Dark-field examinations of these lesions were negative for spirochaetes. Smears showed rare Gram negative bacillus.

PROGRESS.—Healing of the oral lesions was complete in 4 days. After 7 days the lesions of the penis were healed except for slight crusting.

TREATMENT COMPRISED.—(a) Discontinuation of sulfanilamide; (b) force fluids; (c) potassium chlorate mouthwash t. i. d.; (d) calomel powder locally to penis lesions.

IMPRESSION.—This case appears to be a new type of reaction to sulfanilamide therapy, involving the mucous membrane of the mouth, penis, and possibly the eyelid margin. This type of reaction has not been described before, to the author's knowledge, and is presented for record and verification when other cases are observed.

PRACTICAL TREATMENT OF PNEUMONIA WITH SULFAPYRIDINE¹

By Lieutenant (Jr. Gr.) George F. Schmitt, Medical Corps, United States Naval Reserve,² and H. Corwin Hinshaw, M. D.³

Since the advent of sulfapyridine in the treatment of the pneumonias, the average practitioner of medicine has an effective and readily available weapon in hand to use against this common and dangerous group of diseases. It is our purpose to summarize briefly the known facts which are necessary for the intelligent and effective use of this drug.

INDICATIONS FOR CHEMOTHERAPY

The chief indication for sulfapyridine therapy is the diagnosis of pneumonia in the adult patient. The drug should ordinarily be employed whether the infection seems to be mild or severe, for it is impossible to predict that an apparently mild pneumonia will pursue its benign way. Immediately on physical findings of pneumonia or roentgenologic evidence of pulmonary consolidation, associated with

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appropriate symptoms, administration of the drug should be started. A specimen of sputum should be sent to the laboratory for bacteriologic study, but it is usually not necessary to wait for the report. Pneumonia may respond promptly to treatment even though pneumococci are not demonstrated in the sputum. It must also be recalled that pneumococci may be found in the sputum in the absence of pneumonia. The great majority of the cases of lobar pneumonia are caused by members of the pneumococcus group. It must also be recalled that many cases of primary or secondary bronchopneumonia are caused by the same organisms. The distribution of the consolidation is not important.

The drug is not indicated in mild respiratory infections, such as bronchitis, influenza, and so forth, for two reasons: first, the treatment is often not effective and, second, frequently the drug produces symptoms worse than the disease. However, it is easy to imagine circumstances in which the diagnosis of pneumonia cannot be definitely made but in which clinical symptoms are severe enough to justify a trial of chemotherapy.

The question frequently arises as to the use of both serum and sulfapyridine. It can now be said that serum therapy is an adjunct to chemical treatment. Serum treatment plus sulfapyridine seems advisable when bacteremia is present. The prognosis in cases with bacteremia is grave enough to justify the use of all available therapeutic agents. One should consider the advisability of combining serum with sulfapyridine therapy when there is extensive consolidation, in elderly patients and in pregnancy and other conditions which render the outlook grave.

TECHNIC OF ADMINISTRATION

The initial dose is usually large, the exact size depending on the gravity of the situation—age, extent of involvement, presence of bacteremia, pregnancy, and so forth. The initial dose is usually tolerated well, and therefore, one can build up the blood level rapidly before nausea and vomiting intervene. The first dose is usually 30 grains (2 gm.) repeated in 4 hours. Frequently an initial dose of 60 grains (4 gm.) is given and occasionally more.

The surest method of attaining a suitable blood concentration is intravenous administration of sodium sulfapyridine. This preparation has many technical disadvantages and is usually not satisfactory unless the drug is given by mouth also.

In oral therapy, the average adult patient is given 15 grains (1 gm.) every 4 hours until the temperature has been normal for 48 hours or more. Smaller doses may be given for a few more days to prevent the development of a relapse. The drug apparently checks the course

of the disease before immunity can develop. Sometimes no objective benefit is seen in from 48 to 72 hours and in these cases the drug may be discontinued if an adequate blood level has been achieved, particularly if the patient be very uncomfortable. If the blood concentration of sulfapyridine is low and no improvement is demonstrable, the dose may be increased by the oral or intravenous method.

During the administration, it is important to maintain the fluid intake sufficiently high to obtain daily a urinary output of 800 to 1,000 cc. The drug may be precipitated in the renal tubules if insufficient fluids are given. On the other hand, fluids should not be forced too much for fear of facilitating development of pulmonary edema. It is probably advisable to use small doses of sodium bicarbonate with sulfapyridine, for the drug is less easily precipitated in an alkaline urine.

TOXIC PHENOMENA

Nausea and vomiting are frequently experienced. Nearly all patients receiving sulfapyridine are nauseated and a very large percentage may vomit. Many look and feel miserable despite objective evidence of improvement. There are several things one can do about this state of affairs. The most important is to explain the value of the treatment to the patient. It is usually easy to obtain the cooperation of the average patient once it is explained that an upset stomach is a frequent concomitant of the use of this drug. The tablets may be more easily administered by crushing them and suspending in water or milk. They may also be placed under the tongue one at a time (they are tasteless). A popular method to check vomiting is to use the nasal tube, for it is hard to vomit with an empty stomach. The drug, suspended in water, is forced into the tube by means of a syringe and the tube is clamped off for 1 or 2 hours after each dose. After this, siphonage may be instituted by a single bottle suction apparatus or any other suitable method until time for the next dose. The use of the tube is particularly useful in postoperative pneumonia. The inhalation of 100 percent oxygen by means of the B. L. B. mask is frequently of aid in checking vomiting.

The principal disadvantage of the drug is vomiting, but this, although distressing, is not serious in most cases. Vomited doses should be repeated and are often retained.

The drug frequently has some effect on the blood. Agranulocytosis is most feared, but rarely occurs unless the drug is given too long or is given to individuals with true drug idiosyncrasy. It is important to perform a daily white blood cell count. It is best to discontinue treatment if there is a steady drop below normal level particularly if the neutrophils are chiefly affected. A low leukocyte count before treatment is started is not always positive contraindication to use of the drug.

The drug may adversely affect the red corpuscles and for this reason the count of red cells and the concentration of hemoglobin should be determined every 2 or 3 days. We have seen one case of acute hemolytic anemia. Under such conditions the drug must be discontinued and frequent transfusions may be required. The blood count must be kept up regardless of the amount of blood that has to be transfused.

Cyanosis may occur, but it is usually milder and rarer than that due to sulfanilamide. It is not indicative of danger. It is to be remembered that the pneumonia itself may be causing the cyanosis and it is wise to be sure that the patient is receiving sufficient oxygen.

The kidneys may be affected as mentioned previously. Gross hematuria has been rare in our experience because, we believe, an adequate fluid intake is required. One must remember also that a high blood sulfapyridine level may be indicative of impaired renal function with impaired sulfapyridine clearance of the blood.

CONTRAINDICATION

The chief contraindication is a previous major toxic reaction to any of the sulfanilamide series of drugs.

WHAT TO EXPECT FROM THE USE OF THE DRUG

There is usually a prompt fall in temperature but this is probably not a real crisis for there is no new production of immune bodies. There is a slow subjective improvement with a still slower physical and roentgenologic resolution. Apparently the drug arrests the progress of the disease until natural defenses of the body can be mobilized to overcome the infection.

SULFANILAMIDE POISONING

ACUTE HEMOLYTIC ANEMIA

By Captain John T. Bennett, Medical Corps, United States Navy, and Lieutenant Robert L. Ware, Medical Corps, United States Navy

With the increasing use of new therapeutic agents in recent years, the toxic effects of some of the drugs on the cellular elements of the blood have come into prominence. In the case of sulfanilamide, poisoning or idiosyncrasy has been reported to produce, in order of frequency, moderate anemia, excessive leucocytosis, acute hemolytic anemia, neutropenia, and auto-agglutination.

Of these, the most serious appears to be neutropenia. Kracke¹ listed 13 cases, only 3 of which recovered. One fatal case² has been reported in this BULLETIN.

¹ Kracke, R. R.: Relation of drug therapy to neutropenic states, J. A. M. A. 111: 1255, October 1938.

² O'Connell, J. T.: Sulfanilamide poisoning: Report of a fatal case, U. S. Nav. Med. Bull. 36: 61, January 1938.

Excessive leucocytosis occasionally occurs, with the white count rising to 60,000 or above, but no serious after effects have been noted. The two cases of auto-agglutination³ that have been reported were associated with hemolytic anemia, and apparently did not affect the usual progress of the anemia.

A moderate, gradual drop in hemoglobin and red cells appears fairly frequently. A decrease of perhaps 10 percent in the hemoglobin is considered as attributable to the infection treated rather than to the drug⁴ and does not require interruption of therapy. Jaundice does not appear, although there may be urobilinuria.^{5,6,7}

Hemolytic anemia occurred in 21 of Wood's⁸ 522 cases treated with sulfanilamide, compared with only 1 case of neutropenia. These cases occurred at Johns Hopkins Hospital and comprise the largest single series reported. The anemia is evidently due to individual idiosyncrasy to the drug, as there is no demonstrable correlation of the dosage, concentration of the drug in the blood, rapidity of administration, or other feature with the development of the anemia. Some patients take without ill effects four to five times the amount of the drug that produces acute hemolytic anemia in others.

Wood⁸ found the incidence of hemolytic anemia higher in children than in adults; his average incidence for all ages was 4 percent. This is much higher than that observed here, where only one case has developed among nearly 200 patients treated with sulfanilamide. The patients treated in this hospital were, of course, all adult males, chiefly white. The drug was used for a wide variety of conditions by all services.

Other observers also have found the incidence lower than Wood's figure. Among 211 cases of gonorrhea treated with sulfanilamide and reported in this BULLETIN^{9,10,11,12} there was no instance of acute hemolytic anemia. Vest, Harrill, and Colston¹³ had two cases in a

³ Antopol, W., Applebaum, I. and Goldman, L.: Two cases of acute hemolytic anemia with auto-agglutination following sulfanilamide therapy; *J. A. M. A.* **113**: 488, August 1939.

⁴ Bigler, J. A. and Haralambie, J. Q.: Sulfanilamide and related compounds—a review of the literature. *Am. J. Dis. Child.*, **57**: 1110, May 1939.

⁵ Garvin, C. F.: Complications following the administration of sulfanilamide, *J. A. M. A.* **113**: 288, July 1939.

⁶ Kershner, E.: Two cases of anemia following the administration of sulfanilamide, *New Eng. J. Med.*, **218**: 1120, June 1938.

⁷ Jennings, G. H. and Southwell-Sander, G.: Anemia and agranulocytosis during sulfanilamide therapy, *Lancet* **2**: 898, October 1937.

⁸ Wood, W. B.: Anemia during sulfanilamide therapy, *J. A. M. A.* **111**: 1916, November 1938.

⁹ Hering, E. R.: Results of sulfanilamide therapy in gonorrhea, *U. S. Nav. Med. Bull.* **36**: 63, January 1938.

¹⁰ Snowden, R. H. and Bell, R. A.: Sulfanilamide treatment of gonorrhea, *U. S. Nav. Med. Bull.*, **36**: 45, January 1938.

¹¹ Mathis, M. S., and McNamara, P. J.: Gonorrhea treated with sulfanilamide, *U. S. Nav. Med. Bull.*, **37**: 114, January 1939.

¹² Zikmund, A.: Treatment of gonococcal infections with sulfanilamide, *U. S. Nav. Med. Bull.*, **37**: 124, January 1939.

¹³ Vest, S. A., Harrill, H., and Colston, J. A. C.: Use of sulfanilamide in urogenital infections, *J. Urol.* **39**: 196, February 1938.

series of about 300 patients treated, and they cite another series of 400 cases treated without a single instance of hemolytic anemia.

Negroes appear to be affected more frequently than whites.¹⁴ Of 33 case reports^{3 8 13 15 16 17 18} that we have found, 20 were in Negroes and 8 in whites; in 5 cases the race was not indicated. The racial distribution of patients who have received the drug is not known; however, it seems safe to assume, on the basis of relative population, that more whites than Negroes have taken the drug. It thus appears that hemolytic anemia as a complication of sulfanilamide therapy is decidedly more frequent in Negroes than in the white race.

Characteristically, hemolytic anemia develops with dramatic suddenness on from the second to the sixth day of sulfanilamide therapy. It is preceded by nausea and headache that may develop from 6 to 12 hours prior to the sudden hemolysis. Overnight, from one- to two-thirds of the patient's blood is hemolyzed; the red count and hemoglobin fall correspondingly; the white count and the temperature rise; deep jaundice appears within a few hours, with urobilin in the urine, high icterus index, negative direct and positive indirect Vandenberg reactions.

Treatment is aimed at eliminating the drug and restoring the lost blood. Early transfusion is indicated, followed by the usual measures for secondary anemia. The importance of prompt transfusion is emphasized by the fact that in the two reported fatal cases^{15 19} transfusion was not given in one and in the other was given 1 week after the onset of hemolysis.

CASE REPORT

The patient was a Negro mess attendant, aged 25, admitted on September 26, 1939, with acute tonsillitis of 3 days' duration. Physical examination was essentially negative except for the throat. The tonsils were enlarged, acutely inflamed, pitted, with spots of purulent exudate. There was some swelling of the peritonsillar tissues at the upper pole of the right tonsil, but no fluctuation. Temperature on admission was 102°; white blood count, 12,200 with 7 percent young neutrophils, 80 percent mature neutrophils, and 13 percent lymphocytes. Sulfanilamide with equal doses of sodium bicarbonate was started with 20-grain doses four times a day, diminishing the dose daily. A total of 240 grains was given in 96 hours.

The temperature curve gradually fell, reaching normal on the morning of September 29. At this time the throat appeared much improved. The con-

¹⁴ Long, P. H.: Sulfanilamide and its derivatives, *Am. J. Nurs.* **39**: 719, July 1939.

¹⁵ Wood, H.: A fatality from acute hemolytic anemia which developed during the administration of sulfanilamide, *South. M. J.*, **31**: 646, June 1938.

¹⁶ Ottenberg, R.: Clinical experiences with sulfanilamide therapy, *N. J. State Journ. Med.* **30**: 418, March 1939.

¹⁷ Kohn, S. E.: Acute hemolytic anemia during treatment with sulfanilamide, *J. A. M. A.* **109**: 1005, September 1937.

¹⁸ Harvey, A. M., and Janeway, C. A.: Development of acute hemolytic anemia during the administration of sulfanilamide, *J. A. M. A.* **106**: 12, July 1937.

¹⁹ Koletsky, S.: Fatal hemolytic anemia following the administration of sulfanilamide, *J. A. M. A.* **113**: 291, July 1939.

centration of blood sulfanilamide was 5.74 mgms. per 100 cc. of blood; blood count: RBC. 4,190,000; WBC. 9,300, with 72 percent neutrophiles. That afternoon his temperature rose, and on the following morning he appeared acutely ill; he was nauseated, showed a pronounced jaundice, and complained of headache. The icterus index was 55 and the indirect Vandenberg test showed 64 mgms. of bilirubin per 100 cc. of serum; the direct Vandenberg was negative. The urine was of the same port wine color as the blood serum, with more than 100 mgms. of albumin per 100 cc., strongly positive for urobilin and for hemoglobin, loaded with red and white blood cells. The red blood count was $2\frac{1}{2}$ million, white count 28,500 with a strong shift to the left.

Sulfanilamide was discontinued, and an intravenous infusion of dextrose in saline was given. Fluids were forced by mouth, and a hypodermoclysis was given in the afternoon. Ferrous sulphate was started by mouth. On the following day the red count had dropped further and the white count had risen. A transfusion of 500 cc. of whole blood was given. On the next day, October 2, there was obvious improvement, and thereafter his recovery was uneventful.

Moist blood preparations showed no sickling after 24 hours; the fragility of the red cells was within the normal range.

BOOK NOTICES

Publishers submitting books for review are requested to address them as follows:

The Editor,

UNITED STATES NAVAL MEDICAL BULLETIN,
Bureau of Medicine and Surgery, Navy Department,
Washington, D. C.

(For review)

TEXTBOOK OF CLINICAL NEUROLOGY, by *Israel S. Wechsler, M. D., professor of clinical neurology, Columbia University, New York; neurologist, The Mount Sinai Hospital; attending neurologist, Neurological Institute; formerly attending neurologist, The Montefiore Hospital, New York.* Illustrated. W. B. Saunders Co., Philadelphia and London, 1939. Price \$7.

This is the fourth revised edition of a very excellent and well-known volume by an experienced teacher and author. It is intended primarily as a text for students and general practitioners, but can be considered also a valuable addition to the library of any neurologist.

Approximately 4 years have elapsed since the publication of the previous edition, and the author has revised certain chapters, and in general brought this edition up to date in all essential details. The new material which he has incorporated has been properly evaluated and considered to be of permanent value. He has added such material as the smell tests of Elsberg, a description of the newly recognized carotid sinus syndrome, some new data about petrositis, a discussion on the premotor region syndrome, and remarks on the present status of electro-encephalography in relation to epilepsy, and and its possible future value in connection with cortical and subcortical tumors. He has largely rewritten the chapter on neuritis, to conform to the changing concept of that condition, and has made other changes, small but important, too numerous to discuss here.

In general, this publication has maintained the same high standard set by the author's previous editions.

DIABETES by *Edward L. Bortz, A. B., M. D., F. A. C. P., associate professor of medicine, Graduate School of Medicine, University of Pennsylvania;* Second edition, 296 pages, F. A. Davis Co., Philadelphia, publishers, 1940. Price \$2.50

Diabetic manuals have multiplied in the last 20 years for the excellent reason that each doctor or each diabetic clinic, as the number of

diabetic patients has increased, has found it necessary to provide simple and intelligent information regarding diabetes for its patients in order to bring about that ideal cooperation between patient and physician which is actually the foundation of success in the treatment of this disorder.

In this volume the usual topics are presented simply, with numerous illustrations and practical directions which will be found useful for physician and patient alike. A discussion of the nature and the history of diabetes is followed by a discussion of the causes of diabetes. Perhaps the author accepts the influence of nervous and emotional factors as of more frequent and greater importance than most authors, but he properly places the pancreas and its insulin producing function as central in the mechanism of diabetes. The diagnosis of diabetes and other forms of glycosuria is properly stressed. Many pages are given to a discussion of food and dietotherapy. The complications of diabetes including pregnancy, surgery, dentistry, the care of the feet, neuritis, acidosis, are simply described, often with good illustrations and simple directions.

ARTHRITIS, AND ALLIED CONDITIONS, by *Bernard I. Comroe, A. B., M. D., F. A. C. P., instructor in medicine, University of Pennsylvania; ward physician, Hospital of the University of Pennsylvania.* 752 Pages, 200 illustrations. Lea and Febiger, Philadelphia, 1940, Price \$8.50.

The author has in commendable manner assembled the more recent as well as the older ideas, theories and facts regarding arthritis and allied conditions. The book is well printed, easily legible, the sections which the author apparently desires to stress are boxed, thus attracting more than usual attention of the reader. The contents and index are complete. A rather exhaustive discussion is presented of the different types of arthritis, the rheumatoid type, of course, receiving the greater emphasis. The bibliography is extensive.

The book well fulfills the purpose of the author as stated in the preface "to present information concerning arthritis and allied conditions which may be of practical value to the physician in the diagnosis and treatment of his patients." The author lists some 40 diseases which may be mistaken for rheumatoid arthritis. He advances the merits of different forms of treatment such as physiotherapy, massage, foreign proteins, chaulmoogra oil, iontophoresis, bacterial antigens, intravenous vaccines, endocrine preparations, vitamine D, sulphur, gold, bee venom, sulfanilamide, sulfapyridine, vitamine C, vitamine B, and diet. He discusses the role of the tonsils, diseased teeth, accessory nasal sinuses, chronic prostatitis, chronic cholecystitis, and, other chronic infections.

The diagnosis and treatment of low back pain, which is of great importance to the naval medical officer is well discussed. The book is complete, including such subjects as the effect of jaundice on arthritis,

rheumatic fever, gonorrheal arthritis, gout, painful shoulder, painful feet, sciatica, tumors of joints and tendon sheaths, role of allergy in arthritis, etc.

THE PATHOLOGY OF INTERNAL DISEASES, by *William Boyd, M. D., M. R. C. P., Ed., F. R. C. P., London, Dept. Psych., F. R. S. C., professor of pathology and bacteriology, The University of Toronto, Toronto: formerly professor of pathology, The University of Manitoba, Winnipeg, Canada.* Third edition. 874 pages, illustrated. Lea & Febiger, Philadelphia, 1940. Price \$10.

This is a thoroughly revised edition of a book which medical men have known and have liked so well through two previous editions. The author has continued to carry out his original plan of putting out a book of medical pathology covering the diseases found in the medical wards of a large hospital. It not only serves as a pathology but also is a most excellent book on medicine, for the etiology, relation of symptoms to lesions, and laboratory aids to diagnosis of the various diseases are masterfully discussed in a very interesting and easily readable style.

Much new material has been included in the volume and a great deal of that which is retained has been rewritten. Obsolete material has been deleted. Among the many important additions are sections of hypertensive heart disease, vitamin "K" in relation to bleeding in jaundice, the increased frequency of bronchiogenic carcinoma, the reticuloses, equine encephalomyelitis, extrarenal uremia, the relation of pyelonephritis to arterial hypertension, and the prevention of silicosis.

The book is well illustrated with 353 engravings and 4 colored plates. A comprehensive list of references is given after each chapter for those interested in the sources of information or in additional reading.

This book should be read and kept at hand for ready reference by everyone interested in any branch of medicine.

QUANTITY FOOD SERVICE RECIPES. Compiled by the *Administration section of the American Dietetic Association*, under the direction of *Adeline Wood, B. S., chairman.* Illustrations by *Jean McConnell.* Prologue by *Lena F. Cooper, chief, department of nutrition, Montefiore Hospital, New York.* First Edition. 436 pages. Over 1100 recipes. J. B. Lippincott Co., Philadelphia. Price \$4.

This is a recipe book on quantity cooking. It contains 18 chapters on (a) appetizers and relishes; (b) beverages; (c) breads and rolls; (d) cakes; (e) cookies and small cakes; (f) desserts; (g) doughnuts; (h) fish; (i) icings and fillings; (j) luncheon dishes; (k) meats and poultry; (l) pies and pastries; (m) salads; (n) salad dressings; (o) sandwich fillings; (p) sauces; (q) soups; and (r) vegetables.

The compilers claim this to be the first book in which every recipe has been tested and approved by the American Dietetic Association. The recipes in this book are not the work of any one person, but result

of a major project of the American Dietetic Association, assembled under the direction of the supervising dietitian of the Mount Sinai Hospital, New York City.

The recipes have been contributed by dietitians in hospitals, schools, colleges, and restaurants. Every recipe, in addition to the quantities, ingredients, and number of servings, gives complete procedure and in many instances the necessary utensils that may be used to the best advantage.

There is no general index but each chapter is provided with its own table of contents which is very convenient and practical. The three tables: "Tables of Quantities and Yields"; "Approximate or Average Weights of Various Commodities"; and "Number of Servings in Standard Containers" would be of inestimable value to commissary officers and dietitians in issuing provisions to the various galleys, thereby instituting a splendid system of cost control.

This excellent recipe should be a "must" book on every dietitian's desk.

CLINICAL TOXICOLOGY by Clinton H. Thienes, M. D., Ph. D., professor of pharmacology and head of the department of pharmacology, School of Medicine, University of Southern California, Los Angeles; attending pathologist (toxicology), Los Angeles County Hospital. First Edition. 309 pages. Lea & Febiger, Philadelphia, 1940. Price \$3.50.

This is an excellent small manual on toxicology. The various poisons are grouped according to the body tissues affected and the essential clinical aspects are given. In addition, sections dealing with the principles of treatment, symptom diagnosis, and chemical diagnosis are included.

While this book does not, nor is it expected to, rival the larger and more exhaustive works on the subject of toxicology, it is quite surprising that such completeness could be obtained in so little space. It is recommended to students, general practitioners, and as a ready laboratory reference, even to those already possessing larger books on the subject.

VIRUS AND RICKETTSIAL DISEASES, A Symposium Held at the Harvard School of Public Health, June 12-June 17, 1939. Pp. 907, Harvard University Press, Cambridge, Mass. Price \$6.50.

A symposium on virus and rickettsial diseases was held at the Harvard School of Public Health in June 1939. The presentations of the eminent authorities participating have been recorded in the form of 34 separate essays collected and published by the Harvard University Press under the title *Virus and Rickettsial Diseases*.

These essays are remarkable because of the thoroughness with which the essayists have considered previous contributions of a relevant nature. All bring the information concerning their particular

subject right up to the last minute. The collected essays constitute a valuable book of reference. Where necessary, a suitable relationship is maintained between essays so that needless overlapping and repetition is avoided. The bibliographic references are extensive and in keeping with the quality of the essays. For the naval medical officer this volume has particular interest and importance, because of the special significance of virus diseases (a) among young adults, (b) among troops, and (c) in special geographic situations. It should be a part of the library of every medical officer.

In the opinion of the reviewer, who has long maintained a special interest in the problems of viruses and virus diseases, this collection of essays represents one of the most important publications of the past several years, certainly the most important one on virus diseases since the publication of *Filtrable Viruses* by Rivers in 1928. Incidentally, it is of historic interest to note that the somewhat similar collection of essays collected and published by Rivers at that time was the first book on virus diseases to be printed in English.

This publication of the essays of the Harvard symposium can do much to promote among medical men a more workable understanding of the viruses and of the importance of virus diseases. To the civilian physician, the importance of the facts presented therein is great. To the medical officer of our armed forces, it has an even more important significance.

MANUAL OF PERIPHERAL VASCULAR DISORDERS by *David W. Kramer, M. D., F. A. C. P.*, assistant professor of medicine, Jefferson Medical College; assistant physician, Jefferson Hospital; chief clinical assistant vascular clinic, Jefferson Hospital; visiting physician, medical division, Philadelphia General Hospital; consultant on peripheral vascular disorders, Philadelphia General Hospital; attending physician, metabolic division and chief of diabetic clinic, Jewish Hospital; attending physician and chief of diabetic clinic, St. Lukes and Children's Hospital, metabolist to Eagleville Sanatorium. Pp. 448, The Blakiston Co., Philadelphia, Pennsylvania, publishers, 1940. Price \$6.

This is a practical and comprehensive manual of peripheral vascular disorders. The advances in this important field have been numerous and a real need for an up-to-date book such as this has existed for some time.

The arrangement of the subject matter is excellent. The book is divided into four parts. The first part deals with the history, physical signs and symptoms and the numerous special tests used in the study of the peripheral circulation. In the remaining three parts, individual diseases and conditions are considered under the following headings: (a) The occlusive vascular diseases, (b) the vasospastic and the vasodilator disorders, and (c) gangrene and disorders of the veins. The importance of a careful study of the physical signs and symptoms is properly stressed. The various circulatory tests are enumerated,

their technic and clinical application discussed, and their comparative usefulness evaluated. The accepted methods of treatment are outlined in detail and their relative merits discussed. The book is well indexed and a complete bibliography is given at the end of each of the four parts of the book.

This monograph can be highly recommended and will be of practical value to the physician in the study and management of these important disorders.

CLINICAL HEART DISEASE. By *Samuel A. Levine, M. D., F. A. C. P.*, assistant professor of medicine, Harvard Medical School; senior associate in medicine, Peter Bent Brigham Hospital, Boston; consultant cardiologist, Newton Hospital physician, New England Baptist Hospital, Boston. Second edition, revised and reset. W. B. Saunders Co., Philadelphia, 1940. Price \$6.

The first edition of this book appeared 3 years ago and was accorded well merited praise in the BULLETIN's review of January 1937.

The same clear and concise presentation of the subject, that undoubtedly accounted for the success of the first edition, has been adhered to in this edition.

In response to reviews, comments, and criticism, corrections and changes have been made throughout the text and, because of its growing interest and importance, a chapter on the medico-legal aspects of heart disease has been added.

The concluding chapter devoted to clinical electrocardiography, an outstanding feature of the work, has been brought abreast current trends by the adoption of the technic and nomenclature of the precordial lead recommended by the American Heart Association and the Cardiac Society of Great Britain and Ireland.

This book is highly recommended as a practical treatise on clinical heart disease.

CARDIOVASCULAR-RENAL DISEASE by *Lawrence W. Smith, M. D., Edward Weiss, M. D., Walter I. Lillie, M. D., Frank W. Konzelmann, M. D., and Edwin S. Gault, M. D.* D. Appleton-Century Co., New York, publishers, 1940. Price \$4.50.

This small, nicely bound, well-printed book sets forth the clinical manifestations ophthalmological and pathological findings of cardiovascular-renal disease. It is divided into seven parts, namely; hypertensive cardiovascular-renal disease, essential hypertension (nephrosclerosis), senile atherosclerosis (arteriosclerosis), nephritis, nephrosis, clinico-pathological considerations, appendix and bibliography. There are numerous photomicrographs, photographs, drawings, charts and diagrams throughout the book.

The appendix contains some general information, tables, charts, and laboratory procedures relative to cardiovascular-renal disease. It is regrettable that the laboratory procedures show rather serious

errors or omissions. The bibliography contains approximately 350 valuable references.

Medical students and physicians desiring a brief review of cardiovascular-renal disease may find this book useful.

CHEMOTHERAPY AND SERUM THERAPY OF PNEUMONIA, by *Frederick T. Lord, M. D.*, clinical professor of medicine, emeritus, Harvard Medical School; member of the board of consultation, Massachusetts General Hospital, *Elliott S. Robinson, M. D., Ph. D.*, director, division of biologic laboratories, Massachusetts Department of Public Health, and *Roderick Heffron, M. D.*, Medical associate, the Commonwealth Fund; formerly field director, pneumonia study and service, Massachusetts Department of Public Health. 174 pages. The Commonwealth Fund, New York, publishers, 1940. Price \$1.

This little book is the third in a series of handbooks published by the Commonwealth Fund on serum therapy of pneumonia which brings up to date and in addition gives a complete resume of chemotherapy of pneumonia. The problem of when to use serum therapy alone, sulfapyridine alone, or a combination of the two is thoroughly discussed. It gives the indications and contraindications for these various forms of treatment, their relative merit, dosage, methods of use, precautions to be observed, and results obtained.

This little handbook gives a brief and complete summary of the present theory and practice regarding these procedures and should prove a most valuable guide for the therapy of this important disease.

THE DIAGNOSIS AND TREATMENT OF PULMONARY TUBERCULOSIS, by *John B. Hawes, 2d, M. D.*, late president of the Boston Tuberculosis Association; director of the National Tuberculosis Association; former instructor in the graduate school of medicine, Harvard University; and *Moses J. Stone, M. D.*, assistant professor of medicine Boston University School of Medicine; physician to the chest clinic of the Massachusetts Memorial Hospital, Boston, Massachusetts; physician to the tuberculosis clinics of the Boston Health Department; assistant physician Beth Israel Hospital. Second edition, revised by *Dr. Moses J. Stone*, with a foreword by *Richard C. Cabot, M. D.* Pp. 260. illustrated with 75 engravings. Lea & Febiger, Philadelphia, Penna., publishers, 1940. Price \$2.75.

The second edition of this brief and concise textbook on the diagnosis and treatment of pulmonary tuberculosis maintains the general excellence and usefulness of the previous edition.

In keeping with the newer concepts of the prevention and treatment of pulmonary tuberculosis, this section of the book has undergone considerable revision. This is particularly true of the chapter on collapse therapy. A brief chapter on the somewhat controversial subject of allergy and immunity and a chapter on mental aspects of pulmonary tuberculosis have been included in this edition.

While this little book is brief and some sections are somewhat sketchy, it is still a comprehensive manual containing all of the essential data. For those who wish more detailed information, a list of references is appended to each chapter. The compact summary at the end of each chapter is also helpful.

This excellent and authoritative little book should prove to be very valuable to the practitioner.

YOUR CHEST SHOULD BE FLAT, by *S. A. Weisman, M. D., F. A. C. P.*, assistant professor of medicine, University of Minnesota; member of consulting staff in tuberculosis, Glen Lake Sanatorium, Oak Terrace, Minnesota; member of staff, St. Andrew's and St. Mary's Hospital, Minneapolis, Minn. Pp. 145, 17 tables and 74 illus. in 49 figures. J. B. Lippincott Co., Philadelphia, Penna., publishers, 1938. Price \$2.

In this small book the author presents evidence tending to prove that the normal chest is flat and that the deep, narrow chest is more susceptible to tuberculosis. His study is based on chest measurements and calculations of the thoracic index on several hundred healthy university students, several hundred tuberculous patients and over twenty thousand Minneapolis school children. His conclusion that "the deep chest makes better soil for tuberculosis" seems justified.

The book is designed for use by the layman as well as by the physician and should be of practical value to teachers, social workers, public-health officers, and physical-education directors.

CLINICAL DIABETES MELLITUS AND HYPERINSULINISM, by *Russell M. Wilder, M. D., Ph. D., F. A. C. P.*, professor and chief of the department of medicine, The Mayo Foundation for Medical Education & Research, University of Minnesota; head of the section on metabolism therapy, division of medicine, The Mayo Clinic, Rochester, Minn. Pp. 458, illustrated. W. B. Saunders Co., Philadelphia, Penna., publishers, 1940. Price, \$6.

This excellent monograph, well printed and bound, summarizes the clinical consideration and methods which the author in an exceptional experience has found most useful in the diagnosis and treatment of diabetes. The bibliography is extensive and the author has been most generous in giving credit to other workers. Detailed consideration of the theory of carbohydrate metabolism, physiology of experimental diabetes and the pathology of diabetes are purposely omitted.

He introduces the subject by a discussion of the blood sugar and its regulation by a system of opposing forces, chiefly hormones and nerve impulses with some independent action by the liver. While recognizing the physiologic importance of the pituitary body in this connection, he opposes the theories which attribute to the pituitary body much etiologic or pathogenic importance in clinical diabetes. He accepts the unitarian concept of diabetes, defining the disease as an abnormality of metabolism due to insufficiency of the insular activity of the pancreas, or an inadequate insular reserve. As a result the homeostasis of the blood sugar is permanently disturbed. Although other abnormalities exist in diabetes, none is so useful for diagnostic purposes as hyperglycemia. For these clear restatements of fundamental concepts, students of diabetes will be grateful.

In the pathogenesis of diabetes he emphasizes the role of heredity but also accepts the role of climate with a somewhat less critical manner than is displayed elsewhere. Certainly the statistical evidence necessary for a demonstration of differences in diabetic morbidity due to differing climates is as yet unsatisfactory.

Therapy, including the use of diets and insulin as well as the treatment of surgical and other complications, occupies one half the book. Almost all patients at the Mayo Clinic who require insulin receive protamine zinc insulin.

An increase in the operative mortality among diabetics was due to the increasing proportion of operations for carcinoma of the stomach and intestine and the advancing age of the patients. Genito-urinary operations numbered 153. Among 218 cases of acromegaly only 13 cases of frank diabetes (6 percent) were found.

The chapters dealing with hyperinsulinism include case reports of 12 instances of islet cell tumors as well as an excellent summary of the diagnosis and treatment of this important but difficult problem.

OBESITY AND LEANNESS by *Hugo R. Rony, M. D.*, formerly associate in medicine and chief of endocrine clinic, Northwestern University School of Medicine, Chicago, Illinois; formerly attending physician, Cook County Hospital, Chicago, Illinois; member Central Society for Clinical Research, the Association for the Study of Internal Secretions. Pp. 300, illustrated with 32 engravings. Lea & Febiger, Philadelphia, Penna., publishers, 1940. Price \$3.75.

Although obesity has for many years been regarded chiefly as a disturbance of caloric balance, to be treated by dietary means, the emphasis in this book is placed upon aspects of the problem which are usually included in the fields of endocrinology, neurology, cell physiology and genetics. Leanness and obesity are presented as different facets of a single problem, in which the newer methods and knowledge in many fields are applicable.

The reader interested in the fundamental chemical and metabolic background of the problem will find a well-considered summary of recent studies bearing upon the metabolism and transfer of fat. The histology and chemical nature of deposited fat are considered in relation to the curious regional distribution of lipophilia of fat tissue. An excellent discussion of the physiology of hunger and appetite in relation to the caloric balance brings out the peculiar contrast between the effects of different types of hypoglycemia upon hunger, as well as the part played by the thyroid, adrenals, and pituitary. The old belief that women gain weight immoderately at the climacterium is disproven. No support in our present knowledge is found for the theory that abnormal internal secretory activity of the pancreas (hyperinsulinism) is commonly a factor in the general pathogenesis of obesity.

Chapters on the classification and diagnosis of obesity and leanness give excellent clinical descriptions of both common and uncommon

types. Therapy is described under two headings, causal and non-specific, the latter being chiefly dietary. A practical discussion of varied dietary problems, together with the use of drugs, exercise, rest, etc., is included. The book is well written with a properly critical attitude and will be found useful for any practicing physician.

NUTRITION AND DIET IN HEALTH AND DISEASE, by *James S. McLester, M. D.*, professor of medicine, University of Alabama, Birmingham, Alabama. Third edition, revised, 838 pages. W. B. Saunders Co., Philadelphia, publishers, 1939. Price \$8.

Of all the books written on nutrition and diet, the third edition of Dr. McLester's book deserves special commendation. It brings out the problem of nutrition as it is viewed at the present time without neglecting to describe older conceptions.

Throughout the book the author favors some of the more conservative aspects of nutritional treatment of various diseases, although at the same time he quotes recent trends, as for instance, in the management of peptic ulcer or hemorrhage.

An especially interesting chapter is one describing the nutritional factors of lesser importance, stressing "satiety factor" in planning a menu.

It is gratifying to note the author's emphasis on subcaloric diets in diseases of the heart and blood vessels. The reviewer disagrees with Dr. McLester's advice to use agar for constipation because agar is known to interfere with the proper digestion of foods and conditions the gastrocolic reflex of defecation to a definite distention.

The description of low-cost diets is very helpful. The reviewer is puzzled why the author missed the comparative evaluation of digestibility between evaporated and plain milk.

LIFE, HEAT, AND ALTITUDE, Physiological Effects of Hot Climates and Great Heights, by *David Bruce Dill, Fatigue Laboratory, Harvard University*. 211 pages, Harvard University Press, 1938. Price \$2.50.

The contents of this excellent 200-page book comprise 8 Lowell Lectures covering a survey of life in hot climates and at high altitudes. The chapter headings are: (a) Energy exchange and environment, (b) perspiration, (c) thirst, (d) man in hot climates, wet and dry, (e) animal life in deserts, (f) animal life in great heights, (g) man in high altitudes, (h) high altitude flight.

The author is a distinguished physiologist who has summarized basic studies in applied physiology conducted at the Fatigue Laboratory, Harvard University, in the desert around Boulder Dam, and at high altitudes in the Andes.

The material in the book, presenting as it does the advanced concepts of the laboratory in terms of field experience, will be of great value to naval medical officers confronted with problems arising from existence in hot atmospheres and at high altitudes. Of immediate

application are the conclusions with respect to fluid and salt requirements.

The book is comparable to Cannon's *Wisdom of the Body* and L. J. Henderson's *Fitness of the Environment* in presentation of material and it should be included in every naval library.

HUGH YOUNG, *A Surgeon's Autobiography*. 554 pages with over 100 drawings by William P. Didusch and three color prints. Harcourt, Brace & Co., New York, publishers, 1940. Price \$5.

Dr. Young's life is a saga that starts in Texas in 1870, and stretches through 70 years that are so packed with adventures and extraordinary incidents that no dramatics or literary touches are needed to make the book downright fascinating. It has been some years since this reviewer has found a book that could keep him so wide awake and so absorbed in the historical lore of the thing for so many hours on end.

Dr. Young can write more authoritatively than anyone on this history of the development of urology during the past 50 years for the plain reason that he has had more to do than anyone with the making of that history. A surgeon and urologist by profession, Dr. Young is more interested in cold facts than in literary fandangles, and although a prodigious contributor to medical literature for nearly half a century, he still prefers a plain straightforward use of the American language to the affectations of a superscientific tone or the literary embellishments, subtleties, and dramatics that are nauseating enough in ordinary biographical accounts and somehow doubly so in stories with medical settings. If we were to compare Young's story with any other, we should choose that by the late Clarence Darrow, a nonliterary life by a nonliterary man, and yet one who had a mastery of words for the particular use of his particular profession.

The story really starts before 1870, because it reveals the adventurous blood of the father and grandfather Young who were both Confederate generals in the Civil War, the grandfather having served also in the Mexican War. This may explain Dr. Young's great interest in the World War and his allotment of a major portion of the book to an account of his military experiences and urological achievements with the A. E. F.

Young has certainly had more than a natural share of good luck and opportunity, being endowed with mechanical talent and inventiveness, academic inclinations, and the manual dexterity that contributes toward the making of a master surgeon; but his best luck was in being born at a time which permitted him to enter the Johns Hopkins Hospital in the 90's and thus enjoy the untold privileges and inspiration that had to come from close association with such makers of American medicine as Halsted, Osler, Welch, Kelly, Cushing, Flexner, and Finney.

It is somewhat questionable how the lay reader or even the non-urologist doctor will take to the 130 pages devoted to the development of urological operations and urological instruments, even with Young's most skillful treatment in non-technical language, and with William Didusch's incomparable drawings; but for the urologist, if he has the slightest interest in the history of his profession, this section will have an enormous appeal because of the author's intimate connection with nearly all of the major advances in this field since 1900.

The account of Diamond Jim Brady, his illnesses, personal quirks, and his benefactions contains numerous glimpses that have not been recorded elsewhere, and tells the whole story of the origin of the Brady Foundation. Of course the Brady Institute has contributed a tremendous service to individuals afflicted with urological ills but as these benefits scarcely extend beyond the lifetime of the individual patients, such a contribution is as nothing when compared to the benefits it has brought about through research and teaching that will be perpetuated down through the coming generations. Likewise through his talents as a teacher, inventor, organizer, and administrator, Dr. Young has indeed done far greater deeds for his Nation than he has through his direct services to patients.

The part of the book that most intrigued this reviewer was the account of the author's efforts and achievements in his antivenereal program in the American Expeditionary Forces. With Young's knowledge of the subject, his resourcefulness in finding ways of cutting red tape, his ability in mapping out a practical working plan, and very largely with the aid of Pershing's intense interest in and whole-hearted backing of Young's program, the American Army in France established an all-time military record for low venereal incidence—16 cases per 1,000 men per year.

No American urologist should deprive himself of the keen enjoyment to be had in the reading of Hugh Young's story. Any doctor or intelligent layman will come through the book highly entertained and with the certain satisfaction of knowing that he is better informed about many vital biological matters and vignettes of our life and times.

MODERN DERMATOLOGY AND SYPHILOLOGY by *S. William Becker, M. D., associate professor of dermatology and syphilology, Kuppenheimer Foundation, University of Chicago, and Maximillian E. Obermayer, M. D., assistant professor of dermatology and syphilology, Kuppenheimer Foundation, University of Chicago.* 871 pages, 461 illustrations in text, 32 full color plates. J. B. Lippincott Co., Philadelphia, publishers, 1940. Price \$12.

The authors in an effort to combine the specialities of dermatology and syphilology have compiled a volume of 57 chapters and some 850 pages. The text itself is presented in a clear and readable manner. A commendable practice is the introduction of each chapter with a

general discussion, which they call orientation, outlining in general terms the problems to be considered in that particular section. The illustrations are quite good and of sufficient number to well illuminate the text. Black and white plates are supplemented by 32 excellent color prints.

Dermatological conditions are classified and discussed according to their etiological factors. Many conditions peculiar to the tropics and rarely seen in this country are omitted in order to conserve space. Presentation of the diagnostic facts and modes of treatment is especially good. Too much reliance on radiation and other physical-therapy methods not usually available to the general practitioner is not evident. Therapeutic measures recommended are sound, modern, and those which have been successful in the author's hands.

The section on syphilis is clearly presented from the viewpoint of a dermatologist. To the reviewer it seems that undue stress is placed on the differentiation of the various skin manifestations with a tendency to lose sight of the main objects of any luetic infection, that of an early diagnosis and an effective treatment. In the chapter on specific treatment of syphilis, available antiluetic drugs are impartially presented with no mention of the therapeutic indices of the various compounds or recommendations as to their effectiveness. For a modern text too little emphasis is placed on Mapharsen, a drug now universally recognized for its high therapeutic index. The recommended scheme of treatment is somewhat similar to that of the Cooperative Clinical Group, and although considered unnecessarily complicated, cannot be criticized.

From a dermatological standpoint this book can be recommended but it is believed that to qualify also as a general text on syphilology, much need be added in the substance of sound therapeutic principles.

CLINICAL PARASITOLOGY by *Charles Franklin Craig, M. D., M. A., (Hon.), F. A. C. S., F. A. C. P., Col. U. S. Army (Retired), D. S. M. emeritus professor of tropical medicine in the Tulane University of Louisiana, New Orleans, Louisiana, and Earnest Carrol Faust, M. A., Ph. D. professor of parasitology in the department of tropical medicine, Tulane University of Louisiana, New Orleans, Louisiana.* Second edition, illustrated with 244 engravings. Lea & Febiger, Philadelphia, Pa., 1940. Price \$8.50.

It is gratifying to find that although this book is only 3 years old the authors have taken the trouble to revise it. As far as this reviewer can find the revision is well done and the essential features of recent contributions to clinical parasitology are now included in this valuable book.

In general the various subjects are discussed in a clear and very readable style. A definite outline for each parasite is followed throughout the book. This has led to some repetition but has made for quick reference. As in the first edition one finds a paragraph

here and there which could be much improved. For example, the life cycle of *E. histolytica* (p. 46-47) which as the authors state is a comparatively simple one has been made quite complicated and their illustration of same (fig. 1, p. 42) is too incomplete to be anything but confusing. In general the illustrations are well selected and well done. An exception are those of the intestinal amoebae some of which are very poor. For example, fig. 10 on page 71 which is supposed to represent some forms of *E. coli* contains sketches of two cysts, "B" and "C", which are rather characteristic of *E. histolytica*.

On the whole the reviewer considers this book excellent and very practical and a book which seems destined to become increasingly popular in medical schools, laboratories, and clinics.

CLINICAL ROENTGENOLOGY OF THE ALIMENTARY TRACT by Jacob Buckstein M. D., visiting roentgenologist (alimentary tract division), Bellevue Hospital, New York City; consultant in gastro-enterology, Central Islip Hospital, Long Island. 652 pages, illustrated. W. B. Saunders Co., Philadelphia, publishers, 1940. Price \$10.

• This book provides a very welcome addition to the literature of gastro-intestinal roentgenology. The need for a complete and modern survey of this field has been felt for some time and it is not saying too much to state that this volume provides the answer to that need.

The author starts with a brief historical résumé giving due credit to the many pioneers who have contributed to this phase of roentgenology. He then points out the need for both fluoroscopy and radiography coupled with clinical background to accomplish accurate diagnosis especially in the borderline cases. He then plunges into his subject considering it seriatim beginning with the esophagus.

Consideration is complete and meticulous throughout and both anatomical and physiological features are given adequate treatment. Illustrative cases together with excellent and numerous reproductions of radiographs are presented concurrently with the text. About 70 pages are devoted to the esophagus, which fact will indicate the thoroughness of the presentation.

The normal stomach is taken up at length, a most important feature, as it is axiomatic that a good knowledge of the normal is an essential prerequisite to the recognition of pathology. The general routine and technic of examination are well described. Careful study of the rugal folds is emphasized as well as contour study of the filled stomach. The diagnosis of chronic gastritis is treated with good judgment which, in the present state of our knowledge, means conservatively. The author points out that this diagnosis is subject to considerable error in interpretation. In making the diagnosis of gastric ulcer, the author emphasizes the importance of the direct sign, that is, the niche. Both malignant and benign tumors are well considered. Relative to this, it is worthy of note that the author men-

tions the special difficulty that one may encounter in the early diagnosis of scirrhus carcinoma due to the manner in which this type of neoplasm infiltrates the submucosa leaving the mucous membrane free of recognizable abnormalities.

In succeeding chapters, duodenitis, duodenal ulcer, postoperative stomach, idiopathic steatorrhea, the nonspecific granulomatus diseases etc., all receive a complete survey. No significant omissions are noted.

The colon is given the same adequate treatment, again not omitting anatomy and physiology. Careful consideration is given the technic of the barium enema. Especially good is the chapter on ulcerative colitis. There is also a good account of lymphogranuloma as it affects the rectum. Eventration and herniation of the diaphragm are well described and illustrated.

The chapters on the biliary tract are excellent. The author favors oral administration of gall bladder dye for cholecystography and recommends the cumulative or divided dose method in which at least two or three doses of dye are given. He sees no harm in using the method in cases of jaundice and points out that in certain cases it is of value. Cholangiography is also discussed. The use of thorotrast for visualization of the liver and spleen is not recommended. Finally the roentgen diagnosis of diseases of the pancreas is well reviewed.

In conclusion it remains only to repeat that this work appears completely adequate to the subject, reflects sound judgment, is excellently illustrated, and well deserves a place of honor on the book-shelf of every roentgenologist and gastro-enterologist.

DIAGNOSIS AND TREATMENT OF HEAD INJURIES, by *Sidney W. Gross, M. D., F. A. C. S., attending neurosurgeon, Beth Israel Hospital; assoc. neurosurgeon, Morrisania City Hospital; adjunct neurosurgeon, Mt. Sinai Hospital; instructor, New York University, N. Y.; and William Ehrlich, M. D., associate attending neurosurgeon, Newark Beth Israel Hospital; neurosurgeon, Barnett Memorial Hospital, Paterson; consulting neurosurgeon, St. Joseph's Hospital, Paterson, N. J.* Introduction by *Percival Bailey, M. D., Ph. D., professor of neurology and neurosurgery, University of Illinois, Chicago.* 275 pages with 94 illus. Paul, B. Hoeber, Inc., New York, publishers, 1940. Price \$5.

The authors have written this practical book in a concise and easy-to-read manner in an effort to give to the clinician, who is somewhat bewildered by so much controversy concerning the proper treatment of head injury, a practical approach to the problem. The book serves its purpose.

The volume deals with the various types of head injuries, their complications and sequels, and the treatment indicated for each. A simple and practical classification of head injuries is presented. An excellent brief description of the anatomy of the scalp, calvarium, blood and nerve supply, sinuses, divisions of the brain, and the physiology of the intracranial structures is given. The volume deals with

the mechanics of skull fractures and brain injuries, and there is included an excellent discussion of gun-shot wounds of the head. The chapter on methods of examination points out the various neurological signs frequently observed subsequent to head injury and their significance in terms of intracranial pathology. Case histories illustrating the various clinical pictures observed in head injury are included. Operative technic is presented in a step-by-step fashion for the procedures most commonly used in traumatic head surgery: trephine exploration, elevation of depressed fractures, subtemporal decompression, and osteoplastic flap. The indications and the technic of encephalography and ventriculography are presented in detail.

The reviewer believes that more space should have been given to the interpretation of clinical signs following head injury, for only by their precise evaluation will the clinician know whether or not an operation is indicated. It is noted that no mention is made of the value of careful and repeated recording of the pulse pressure which in the reviewer's experience is a valuable clinical aid for determining the true state of intracranial pathology. The controversial subject of the value of lumbar puncture in the management of head injury is discussed but it is doubtful if the practitioner with little experience with treatment of head injury will know whether or not to perform such a procedure after reading this volume. The dehydration method of treatment is discussed with several warnings as to the dangers from excessive dehydration. It is believed, however, that more emphasis should have been placed on the complications which develop from giving patients with head trauma too much fluid. No mention is made of subdural hydroma or hygroma which is not an infrequent complication of craniocerebral trauma.

The book is handsomely illustrated and contains a very complete bibliography. On the whole the volume covers the subject of head injury well and gives in a practical manner valuable aid to the clinician faced with the treatment and management of patients with intracranial injury.

A SYNOPSIS OF REGIONAL ANATOMY by *T. B. Johnston, M. D., professor of anatomy, University of London, Guy's Hospital Medical School.* Fourth edition, 1939. With 17 illustrations. 462 pages. Lea & Febiger, Philadelphia. Price \$4.50.

This is a dissecting manual first published in 1921 and the fact that this is a fourth edition nearly 20 years later speaks for its excellence. There are but few illustrations, it being the author's idea that the student should use his own dissection for the illustration.

THE DIVISION OF PREVENTIVE MEDICINE

Commander C. S. Stephenson, Medical Corps, United States Navy, in charge

TOXIC EFFECTS OF ARSENICAL COMPOUNDS

AS EMPLOYED IN THE TREATMENT OF DISEASES IN THE UNITED STATES NAVY, 1939

By Commander C. S. Stephenson, Medical Corps, United States Navy, Chief Pharmacist's Mate, W. M. Chambers, United States Navy, and Mrs. Laura T. Anderson, Senior Clerk, Bureau of Medicine and Surgery

Since November 1924 medical officers of the Navy have been required to make monthly reports of the number of doses of arsenicals administered and a separate report of each case in which ill effects are noted. During the 15 years, 1925-39, in which this information has been compiled, 1,562,265 doses of arsenicals have been administered and 1,063 reactions have been reported.

Previous articles dealing with the information obtained from these reports have been published in the following issues of this BULLETIN:

September 1925	October 1934	January 1938
January 1927	January 1935	October 1938
January 1929	October 1935	January 1939
July 1930	January 1936	October 1939
October 1931	October 1936	January 1940
October 1932	January 1937	October 1940
October 1933	October 1937	

Cases of arsenical dermatitis occurring during the year 1939 were published in the October 1940 issue.

The present article includes all cases, except arsenical dermatitis, which were reported during the year 1939. Comparative figures from the experience of previous years are also presented.

TABLE 1.—*Arsenical reactions, 1939*

Classification	Neocarsphenamine and mapharsen			
	Mild	Severe	Fatal	Total
Arsenical dermatitis *	12	9	1	22
Vasomotor phenomena	12	1	0	13
Blood dyscrasias	1	2	2	5
Liver damage	1	4	1	6
Jarisch Herxheimer	3	0	0	3
Gastro-intestinal	1	0	0	1
Total	30	16	4	50

* Case histories were published in the October 1940 number of the bulletin. 14 of the above reactions (9 mild, 5 severe) were caused by mapharsen.

TABLE 2.—Arsenicals administered during the year 1939 for all diseases

Drug	Doses (grams)				
	0.9 to 3	0.9	0.6 to 0.9	Less than 0.6	Total
Bismarsen:					
Navy.....	0	0	0	218	218
All others.....	0	0	0	239	239
Maparsen:					
Navy.....	0	0	0	58,999	58,999
All others.....	0	0	0	6,400	6,400
Neosarsphenamine:					
Navy.....	0	29	19,397	17,017	36,443
All others.....	0	193	3,223	17,517	20,933
Sulfarsphenamine:					
Navy.....	0	0	15	8	23
All others.....	0	0	56	864	920
Tryparsamide:					
Navy.....	3,306	0	0	3	3,309
All others.....	1,806	0	2	3	1,811
Total.....	5,112	222	22,693	101,268	129,295

TABLE 3.—Arsenicals administered during the 8-year period, 1932-39, for all diseases

Drug	Doses (grams)				
	0.9 to 3	0.9	0.6 to 0.9	Less than 0.6	Total
Acetarsonic:					
Navy.....	0	0	0	166	166
All others.....	0	0	76	729	805
Arsphenamine:					
Navy.....	0	0	149	10,297	10,446
All others.....	0	0	7	706	713
Bismarsen:					
Navy.....	0	0	0	1,762	1,762
All others.....	0	0	1	1,083	1,084
Maparsen: *					
Navy.....	0	0	0	107,610	107,610
All others.....	0	0	0	14,078	14,078
Neosarsphenamine:					
Navy.....	0	5,120	274,339	369,122	648,581
All others.....	0	630	35,714	121,482	157,776
Silver arsphenamine:					
Navy.....	0	0	0	350	350
All others.....	0	0	0	204	204
Sulfarsphenamine:					
Navy.....	0	18	311	7,424	7,753
All others.....	0	7	290	12,267	12,564
Tryparsamide:					
Navy.....	29,431	0	0	13	29,444
All others.....	13,989	0	2	8	13,999
Total.....	43,420	5,775	310,889	647,251	1,007,335

* First administered in 1935.

TABLE 4.—Deaths and severe reactions, following administration of 1,301,913 doses of neoarsphenamine, 1925-39; ratio of deaths and severe reactions to doses

Classification	Deaths		Severe reactions		Deaths and severe reactions	
	Number	Ratio to doses 1 to—	Number	Ratio to doses 1 to—	Number	Ratio to doses 1 to—
Hemorrhagic encephalitis.....	16	81,370	1	1,301,913	17	76,583
Arsenical dermatitis.....	13	100,147	193	6,746	206	6,320
Vasomotor phenomena.....	6	216,986	57	22,841	63	20,665
Blood dyscrasias.....	8	162,739	19	68,522	27	48,219
Acute renal damage.....	2	650,957	5	260,383	7	185,988
Acute yellow atrophy of the liver.....	2	650,957	0	-----	2	650,957
Vascular damage (probable renal hemorrhage).....	1	1,301,913	0	-----	1	1,301,913
Liver damage.....	1	1,301,913	21	61,996	22	59,178
Jarisch Herxheimer.....	0	-----	2	650,957	2	650,957
Gastro-intestinal.....	0	-----	3	433,971	3	433,971
Polyneuritis.....	0	-----	1	1,301,913	1	1,301,913
Border-line hemorrhagic encephalitis.....	0	-----	1	1,301,913	1	1,301,913
Arsenical neuritis.....	0	-----	1	1,301,913	1	1,301,913
Optic neuritis.....	0	-----	1	1,301,913	1	1,301,913
Total.....	49	26,570	305	4,269	354	3,678

TABLE 5.—Deaths following administration of arsenical compounds, 1919-39

Year	Arsphen-amine	Neo-arsphen-amine	Total	Year	Arsphen-amine	Neo-arsphen-amine	Total
1919.....	3	0	3	1931.....	0	0	0
1920.....	1	1	2	1932.....	0	4	4
1921.....	3	1	4	1933.....	0	7	7
1922.....	0	4	4	1934.....	0	3	3
1923.....	0	1	1	1935.....	0	2	2
1924.....	1	2	3	1936.....	0	3	3
1925.....	0	2	2	1937.....	0	1	1
1926.....	0	4	4	1938.....	0	3	3
1927.....	1	4	5	1939.....	0	4	4
1928.....	0	6	6				
1929.....	0	3	3	Total.....	9	58	67
1930.....	0	3	3				

NUMBER OF PERSONS TREATED FOR SYPHILIS AND OTHER DISEASES

Annually on December 31 each activity reports to the Bureau of Medicine and Surgery, on NMS-Form A, the number of persons in that command who have a history of syphilis, and the number of those in the command who were treated during the year with an arsenical compound, heavy metal, or other antiluetic treatment. A report of the number of persons who were treated during the year with an arsenical compound for a disease other than syphilis is also required. This census does not take into account individuals who left the service during the year.

In the following table, treatment data are listed separately for active service personnel and all others. The term *All others* includes Veterans' Administration patients, dependents of naval personnel, retired naval personnel, and native populations of insular possessions.

TABLE 6.—*Syphilis and arsenicals, U. S. Navy, 1939*

Item	Persons		
	Navy and Marine Corps	All others	Total
Strength, Dec. 31, 1939.....	161,736		161,736
Syphilis census, Dec. 31, 1939.....	15,322		15,322
Number of persons treated for syphilis with—			
Arsenicals:			
Bismarsen.....	19	13	32
Mapharsen.....	3,622	456	4,078
Neoarsphenamine.....	2,635	454	3,089
Sulfarsphenamine.....	4	68	72
Tryparsamide.....	201	144	345
Total persons treated with arsenicals.....	6,481	1,135	7,616
Heavy metal compounds:			
Bismuth.....	5,281	955	6,236
Mercury.....	81	7	88
Mixed treatment (specific mixture, etc.).....	4	20	24
Potassium iodide.....	102	16	118
Total persons treated with heavy metal compounds.....	5,468	998	6,466
Number of persons treated for disease other than syphilis with—			
Arsenicals:			
Mapharsen.....	124	0	124
Neoarsphenamine.....	205	1,584	1,789
Sulfarsphenamine.....	0	4	4
Fowler's solution.....	29	1	30
Total persons treated with arsenicals.....	358	1,589	1,947
Heavy metal compounds:			
Bismuth.....	1	138	139
Mercury.....	1	0	1
Total persons treated with heavy metal compounds.....	2	138	140

In table 6 it will be noted that 358 persons in the Navy and Marine Corps were treated with arsenical compounds during the year 1939 for diseases other than syphilis, as follows: Vincent's infection, 313; acne, 6; yaws, 2; and other diseases and conditions, 37.

Of the 1,589 persons in the group all others treated for diseases other than syphilis, 1,586 were treated for yaws and 3 were treated for other diseases and conditions.

VASOMOTOR PHENOMENA

NEOARSPHENAMINE—(23-1939).—This patient, exposed to infection on February 20, 1934, developed an ulcer on the penis which was positive for *Treponema pallidum*.

From February 23 to November 21, 1934, he received 15 injections of neoarsphenamine (amount not recorded) and from March 17 to August 9, 1934, he received 15 injections of bismuth salicylate, a total of 15 grams. The patient stated that he experienced a reaction following the last injection of neoarsphenamine.

A 0.03-gram injection of mapharsen was administered on November 22, 1939, followed by a 0.3-gram injection of neoarsphenamine on November 29, a 0.45-gram injection on December 21, and a 0.6-gram injection on December 29. Immediately after the last injection of neoarsphenamine the patient's face, neck, and shoulders became very red and he became dizzy and vomited. Pulse was faint and rapid and the skin was cold and clammy. The temperature reached 100.8° F. during the day but subsided in the evening after profuse sweating. He

was given 12 minims of adrenalin subcutaneously 2 minutes after the reaction occurred. Recovery in 1 day.

(24-1939).—After exposure to infection on January 20, 1939, this patient developed a primary lesion on the glans penis which was positive for *Treponema pallidum*.

From January 29 to August 9, 1939, he received 16 injections of neoarsphenamine, a total of 7.50 grams, and from March 26 to November 8, 1939, 24 injections of bismuth subsalicylate, a total of 3.27 grams.

The third course of arsenical treatment began on November 15, 1939, with a 0.6-gram injection of neoarsphenamine, followed by 0.6-gram injections on November 22 and 29. Five minutes after the last injection the skin was flushed, the conjunctival blood vessels were injected, and the patient became nauseated and vomited. Several hours later he complained of headache; and temperature of 100.5° F. and pulse 105 were recorded. Both subsided by evening.

Ten minims of adrenalin were given immediately after the administration of the neoarsphenamine, and 5 minutes later 1 gram of sodium thiosulphate was given intravenously. Symptoms rapidly subsided and the patient was returned to duty 1 day after onset of the first symptoms.

(25-1939).—One month after exposure to infection on January 25, 1939, this patient developed a penile lesion, darkfield examinations of which were repeatedly positive for *Treponema pallidum*. A Kahn blood test was 4 plus.

Antiluetic treatment began on March 11, 1939, with a 0.3-gram injection of neoarsphenamine and a 0.1-gram injection of bismuth subsalicylate.

Nine hours after the injection of neoarsphenamine the patient developed a delayed nitroid reaction as manifested by sudden collapse, high fever, headache, rapid pulse, and severe prostration. He was given a 1-gram intravenous injection of sodium thiosulphate. Recovery in 1 hour from onset of symptoms.

(26-1939).—After exposure to infection on March 11, 1939, this patient developed a penile lesion. A Kahn blood test was positive.

Antiluetic treatment began with 0.3-gram injections of neoarsphenamine on March 15 and 22. Ten hours after the last injection the patient suddenly developed a severe chill and a temperature of 104° F. The temperature fluctuated between 100° F. and 102° F. on the following day and returned to normal with no other symptomatology on the third day.

On March 27 a 0.02-gram injection of mapharsen was given with no evidence of a reaction. Recovery in 2 days.

(27-1939).—This patient was given a diagnosis of syphilis in January 1934, because of a positive darkfield examination and positive Kahn blood tests.

From January 20, 1934, to February 4, 1939, he received 19 injections of neoarsphenamine, 44 injections of mapharsen, and 94 injections of bismuth compound as concurrent treatment.

The sixth course of arsenical treatment began with a 0.3-gram injection of neoarsphenamine on March 30, 1939, followed by 0.6-gram injections on April 6 and 13. The reaction occurred immediately after the last injection as manifested by rapid pulse, pain in the lumbar region, consciousness of heart action, slight dizziness, and weakness. The face was flushed and conjunctiva somewhat injected.

The patient was given 1 gram of sodium thiosulphate intravenously and 1 cc. of adrenalin subcutaneously. The symptoms subsided rapidly and recovery occurred in 1 hour.

(28-1939).—This patient was exposed to infection on September 7, 1926, and 1 month later developed a penile lesion which was positive for *Treponema pallidum*.

He received the following arsenical treatment: From October 6 to October 28,

1926, 13.5 grams of neoarsphenamine (number of injections not recorded); from February 26, 1927, to February 15, 1931, 18 injections of salvarsan; from May 26 to July 31, 1934, 8 injections of tryparsamide; and from September 28 to December 28, 1938, 3 injections of neoarsphenamine. He received 127 injections of a bismuth compound and 30 mercury injections as concurrent treatment.

The patient received a 0.3-gram injection of neoarsphenamine on July 20, 1939, and developed a slight chill, fever, and vomiting 10 minutes later. Temperature 101.5° F.; WBC. 13,200

One gram of sodium thiosulphate was given intravenously. Complete recovery occurred in 7 hours.

(29-1939).—A penile lesion and general adenopathy developed after exposure to infection on January 1, 1939. A darkfield examination of the lesion was positive for *Treponema pallidum*.

A 0.3-gram injection of neoarsphenamine was administered on February 11 and a 0.6-gram injection on February 16. Bismuth salicylate was given as concurrent treatment.

Two hours after the last injection of neoarsphenamine the patient complained of feeling chilly, dizzy, nauseated, and weak, and 2 days later a maculo-erythematous rash appeared over the entire body. The throat, pharynx, and conjunctiva were injected. Temperature 104° F.; pulse 100.

On February 21 the patient still appeared extremely toxic with marked gastrointestinal upset and a great deal of emesis. Normal saline was administered intravenously. The symptoms gradually subsided and the skin conditions cleared after 3 weeks' treatment.

The patient was given a 0.02-gram injection of mapharsen on March 16 and a 0.01 gram injection on March 24 without signs or symptoms of further reaction. Recovery in 43 days.

(30-1939).—This patient acquired syphilis in November 1932, and received 30 injections of neoarsphenamine and 52 injections of bismuth up to and including June 24, 1936. Kahn blood tests during this period were negative and the patient was considered cured.

A second syphilitic infection occurred 5 days after exposure on May 20, 1939, when the patient developed a penile lesion which was positive for *Treponema pallidum*.

A 0.3-gram injection of neoarsphenamine was administered on May 25 and a 0.45-gram injection on June 1, 1939. One injection of thiobismol was given as concurrent treatment. Two hours after the injection of neoarsphenamine on June 1 the patient complained of nausea and vomiting. Examination revealed flushed face, injected conjunctiva, temperature 102° F., pulse 130, respirations 20, and WBC. 17,550. Two grams of sodium thiosulphate were given daily. Recovery in 4 days.

MAPHARSEN—(31-1939).—A patient, exposed to infection on January 12, 1929, was given a diagnosis of syphilis because of serological findings.

He received 44 injections of neoarsphenamine and 3 injections of mapharsen from January 13, 1929, to May 16, 1939; 30 injections of mercury from October 19 to December 15, 1932; and 34 injections of bismuth compound from February 15, 1933, to March 29, 1939.

The sixth course of arsenical treatment (five injections of mapharsen, a total of 0.63 gram) was administered between April 19 and May 16, 1939. The patient experienced a slight burning during the administration of the last injection of mapharsen (0.05 gram) and within 5 minutes he complained of severe pain, weakness, and nausea. Two hours later his arm was red and swollen.

The induration gradually subsided without suppuration or sloughing. Recovery occurred in 8 days.

(32-1939).—A patient, exposed to infection April 10, 1939, developed an ulcerative lesion on corona of penis and inguinal adenopathy 1 month later. A darkfield examination was positive for *Treponema pallidum*.

Arsenical treatment was instituted on May 17, 1939, with a 0.06-gram injection of mapharsen. Six hours after the injection moderate chills, slight headache, and a temperature of 103.4° F. were noted. Symptoms rapidly subsided without specific treatment. Recovery in 1 day.

(33-1939).—The source of infection in this case is unknown. The patient was given a diagnosis of syphilis because of 4-plus Kahn blood tests. A preparatory course of 8 injections of bismuth, a total of 1.56 grams, was administered from May 30 to August 2, 1939.

Arsenical treatment began on July 25, 1939, with a 0.03-gram injection of mapharsen. There were no apparent ill effects following this injection. On August 2 he received a 0.06-gram injection of mapharsen and 3 hours later developed a headache, general malaise, and high fever. One gram of sodium thiosulphate was administered on August 2 and 3. Recovery in 5 days.

(34-1939).—After exposure on March 19, 1939, this patient developed two small ulcers on the penis which were positive for *Treponema pallidum*.

From May 19 to October 10, 1939, the patient received 20 injections of mapharsen, a total of 1.04 grams, and 24 injections of bismuth salicylate as concurrent treatment. The third course of arsenical treatment began on October 31, 1939, with a 0.03-gram injection of mapharsen, followed by 0.06-gram injections on November 7, 14, 21, 28, December 5, 12, and 19.

Twelve hours after the last injection of mapharsen the patient suddenly experienced malaise, headache, chills, and fever. Temperature 103° F., pulse 120, respirations 20, and urinalysis showed a very faint trace of albumin. Symptoms gradually subsided under treatment. Recovery in 2 days.

(35-1939).—This patient developed bilateral inguinal adenopathy and secondary macular rash following exposure to infection in February 1938. A Kahn blood test was 4 plus.

From April 1 to June 8, 1938, he received 34 injections of mapharsen, a total of 1.89 grams, and 40 injections of a bismuth compound; and from October 5 to November 30, 1939, 8 injections of mapharsen, a total of 0.48 gram. Four days after the last injection of mapharsen the patient reported to the sick bay complaining of chills and fever. He stated that he experienced chills and fever 6 hours after the last injection. Examination revealed the patient to be pale, weak, and sweating. Temperature 104° F.; RBC, 3,840,000; WBC, 16,100; bands, 10; segs. 68; lymphs. 20; and basos. 2. Treatment consisted of rest in bed, soft diet, and forced fluids. No specific treatment was administered. Recovery in 16 days.

GASTRO-INTESTINAL

NEOARSPHENAMINE.—(36-1939).—This patient, exposed to infection on August 25, 1939, developed a penile lesion which was positive for *Treponema pallidum*.

A 0.3-gram injection of neoarsphenamine was administered on September 9, 1939, and a 0.45-gram injection on September 12.

Ninety-six hours after the last injection the patient developed a headache, general malaise, diarrhea, and general macular nonitching rash. Temperature 104° F.; RBC, 4,730,000; WBC, 10,350; bands, 16; segs. 59; lymphs. 21; and

monos. 4. He was given 1 gram of sodium thiosulphate intravenously on September 19 and 20. Recovery in 7 days.

JARISCH HERXHEIMER

NEOARSPHENAMINE.—(37-1939).—This patient developed a penile lesion 2 months after exposure to infection on January 14, 1939. A Kahn blood test was 4 plus. He received a 0.3-gram injection of neoarsphenamine on March 22, 1939. Ten hours later the patient developed a typical Herxheimer reaction with accentuated symptoms. This condition subsided rapidly under symptomatic treatment. Recovery occurred in 12 hours.

(38-1939).—After exposure to infection in February 1939, this patient developed a typical chancre and macular secondaries. Repeated Kahn blood tests were 4 plus. Antisyphilitic treatment began with a 0.3-gram injection of neoarsphenamine on March 15, 1939.

Twelve hours after the injection of neoarsphenamine the patient developed a Herxheimer reaction with accentuated luetic symptoms. Temperature 104° F. All symptoms subsided without specific treatment. Recovery in 8 hours.

MAPHARSEN.—(39-1939).—Two weeks after exposure to infection this patient developed a penile lesion which was positive for *Treponema pallidum*.

Antiluetic treatment was instituted with a 0.03-gram injection of mapharsen on January 16, 1939. Ten hours after the injection the patient developed a typical Herxheimer reaction. There was marked edema of the local lesions and prepuce, also swelling and tenderness of the inguinal glands. Temperature 103° F., pulse 110. The symptoms subsided completely 3 days after onset. A second injection of mapharsen was given on January 19, with no evidence of a reaction. Recovery occurred in 4 days.

LIVER DAMAGE

NEOARSPHENAMINE.—(40-1939).—The source of infection in this case is unknown. The diagnosis of syphilis was established by positive Kahn blood tests. Arsenical treatment began on July 27, 1939, with a 0.3-gram injection of neoarsphenamine, followed by 0.45-gram injections on August 3, 12, 17. The patient states that he experienced chills, fever, and abdominal cramps the day following the last injection of neoarsphenamine. This condition was not reported until 3 days later, when an examination revealed the sclera and skin to be deeply jaundiced. The stool was clay colored and the urine was greatly discolored by bile. No skin rash was apparent but there was a slight scaling of the skin of the hands. Three 1-gram injections of sodium thiosulphate were administered intravenously. The temperature became normal 3 days after onset of symptoms. Recovery in 3 days.

(41-1939).—After repeated exposures to infection this patient developed a penile lesion on or about March 20, 1939, while on leave and a civilian physician informed him that the lesion was a tropical ulcer. The physician made one darkfield examination which was negative and administered two injections of an arsenical compound. When the patient returned from leave a diagnosis of syphilis was established by secondaries, a 4-plus Kahn blood test, and lesions on mucous surface of foreskin and erosions around meatus.

From April 18 to June 1, 1939, he received 8 injections of neoarsphenamine, a total of 4.35 grams; from August 7 to October 19, 7 injections of mapharsen, a total of 0.42 gram; and 14 injections of a bismuth compound as concurrent treatment.

The second course of arsenical treatment began on October 26, 1939, with a 0.6-gram injection of neoarsphenamine, followed by 0.6-gram injections on

November 3, 10, 17, and 24. Five days after the last injection the patient was admitted to the sick list complaining of nausea, vomiting, yellow skin, weakness, and dull epigastric pains. He stated that these symptoms had been present for 3 days but were relieved by vomiting and he did not report for treatment.

Urinalysis was positive for bile (3 plus); RBC. 4,230,000; WBC. 6,150; bands 8; segs. 53; lymphs. 32; monos. 4; and icterus index 25.

The patient was given 1 gram of calcium levulinate intravenously and daily 1-gram injections of sodium thiosulphate intravenously.

The jaundice gradually disappeared under treatment. Recovery occurred in 26 days.

(42-1939).—This patient, exposed to infection on September 27, 1939, developed a chancre on coronal sulcus. A darkfield examination was positive for *Treponema pallidum*.

Arsenical treatment began with 0.3-gram injections of neoarsphenamine on October 10 and 17, 1939, and three injections, 0.13 gram each, of bismuth subsalicylate as concurrent treatment. Twenty-four hours after the last injection of neoarsphenamine the patient complained of general malaise and drowsiness. The temperature rose to 104° F. Frequent urination and nocturia were noted. On the morning of October 20 the patient's face was markedly edematous; no other edema noted. There was no evidence of itching skin or exfoliation.

October 23: WBC. 13,000; lymphs. 11; monos. 11; bands 14; eosins. 4; segs. 60.

October 25: An icterus tinge was noticeable, and during the ensuing days progressive jaundice developed involving the entire body.

October 26: Urine negative for albumin, positive for bile. Icterus index, 30; VDB, direct action immediate; quantitative, 4.8 mgms.

November 4: Urine positive for bile; WBC. 6,400; lymphs. 16; monos. 12; bands. 2; segs. 70.

November 9: Icterus index, 80; VDB, direct action immediate; 9 mgms. bilirubin per 100 cc. of blood. WBC. 7,900; RBC. 3,950,000; lymphs. 28; monos. 8; bands 3; eosins. 2; segs. 59.

Treatment: (1) 50 cc. of dextrose (50%) each morning with 10 units plain insulin and 1,000 cc. dextrose (10%) with 10 units plain insulin each evening from October 31 to November 13; (2) calcium gluconate 10 cc. intravenously in the morning on alternate days; (3) sodium thiosulphate 10 cc. intravenously in the morning on alternate days; (4) sodium bicarbonate, 15 grains, t. i. d., orally for period from October 31 to November 4; (5) biliary drainage with a duodenal tube and 80 cc. of 33% solution of magnesium sulphate each morning from November 2 to November 4, with no results; (6) intramuscular injections of 1 cc. of liver extract in the morning from November 8 to November 16.

The patient showed marked improvement under treatment and was returned to duty in 54 days apparently fully recovered.

(43-1939).—This patient was exposed to infection on April 1, 1939. Three weeks later a penile sore developed which was positive for *Treponema pallidum*.

He was given a 0.3-gram injection of neoarsphenamine on May 17, and 8 hours later complained of general malaise, fever, severe frontal headache, and pain under the right costal margin. Recovery from this reaction occurred in 24 hours. On May 20 a 0.45-gram injection of neoarsphenamine was administered and a similar reaction occurred, followed in 36 hours by pain and tenderness in the right hypochondrium. Temperature 99° F., pulse 112, and respirations 18. The edge of the liver was palpated 1 inch below the costal margin and quite tender. There was no nausea or vomiting. Systemic symptoms subsided in 3 days but the pain persisted in the liver region.

A 0.02-gram injection of mapharsen was administered on June 1 and no ill effects were noted. Treatment with mapharsen continued and from June 1 to August 2, the patient received 11 injections with no evidence of a reaction. It is believed that the original symptoms were due to a hepatitis which was possibly influenced by the antisyphilitic treatment. Recovery in 73 days.

MAPHARSEN—(44-1939).—This patient was exposed to infection on February 19, 1939, and 1 month later developed a penile lesion which was positive for *Treponema pallidum*.

Arsenical treatment began with 0.03-gram injections of mapharsen on March 20 and March 24 and 0.06-gram injections on March 28 and April 4. Two injections of iodobismutol were given as concurrent treatment.

Twenty-four hours after the last injection of mapharsen the patient developed jaundice, anorexia, slight nausea, bile in the urine, and an icteric index of 8-15. April 17: WBC. 9,600.

One vial of liver extract was administered 3 times daily for a period of 2 weeks. He received six 1-gram injections of sodium thiosulphate intravenously. Symptoms gradually subsided and recovery was complete 70 days after onset of first symptoms.

BLOOD DYSCRASIAS

NEOARSPHENAMINE—(45-1939).—A patient, exposed to infection May 22, 1939, developed a chancre on the penis which was positive for *Treponema pallidum*.

Arsenical treatment began with a 0.3-gram injection of neoarsphenamine on June 22, and continued with 0.45-gram injections on June 26, 28, July 1 and a 0.6-gram on July 8. He received 5 injections of bismuth salicylate, a total of 0.65 gram, as concurrent treatment.

Six days after the last injection of neoarsphenamine the patient complained of sore throat, dysphagia, nausea, and vomiting. Examination revealed the throat to be moderately injected, swollen, indurated, and tender in the submental areas. The skin was flushed and hot and there was a red splotchy rash over the trunk.

Blood picture

Date	RBC	WBC	Hgb.	Juvs.	Bands.	Segs.	Lymphs.	Eosins.	Monos.	Basos.
1939										
July 14.....		3,500			3	6	64	7	19	
July 15.....	4,530,000	3,900	84		5	1	39	7	37	1
July 18.....	5,820,000	4,250	90		9	8	25	1	45	2
July 19.....	5,110,000	6,850	90		11	15	44	4	26	
July 20.....	5,600,000	7,850	90		17	17	31	2	32	1
July 21.....	4,850,000	5,750	94	1	17	34	22	2	24	
July 22.....	4,500,000	8,000	96	3	19	44	24	2	8	
July 23.....	4,900,000	6,400	100		10	52	22	2	12	
July 25.....	4,500,000	5,000	90		14	44	23	6	13	
July 26.....	5,300,000	5,500	90		12	44	28	2	14	
July 27.....	4,500,000	5,400	90	1	8	35	36	2	17	
July 28.....	4,800,000	2,800	87		10	47	33	1	9	
July 29.....	5,350,000	4,200	94		2	34	46	4	10	4
July 31.....	5,450,000	4,600	90		5	50	41		4	

Ten cubic centimeters of pentnucleotide were given twice daily for 2 days and daily for 5 days. His condition steadily improved under treatment, and except for slight fluctuation of the white blood count he was considered satisfactorily recovered in 14 days from onset of symptoms.

(46-1939).—A patient, exposed to infection April 19, 1939, was given a diagnosis of syphilis because of positive Kahn blood tests.

Arsenical treatment began with a 0.45-gram injection of neoarsphenamine on May 20, followed by 0.6-gram injections on May 23, 27, June 3 and 10, a total of 2.85 grams. Reaction resulted from no single injection. Granulocytopenia developed 21 days after the first injection of neoarsphenamine.

A 10-day course of sulfanilamide was instituted on May 28 for the treatment of bubo, inguinal. Treatments were tolerated without reaction.

On June 10, the date of the last injection, the patient had 2 attacks of epistaxis.

Blood Picture

Date	RBC	WBC	Hgb.	Juv.	Myelos.	Bands.	Segs.	Lymphs.	Eosins.	Basos.	Monos.	Plates.
1939												
June 9.....	3,900,000	7,200	77	4	1	8	4	52	3	3	26	-----
June 10.....	3,900,000	6,750	72	1	-----	2	-----	52	3	1	41	-----
June 11.....	3,130,000	5,600	-----	-----	-----	4	1	51	4	-----	40	109,000
June 12.....	4,240,000	-----	84	-----	1	10	2	46	3	2	36	129,000
June 13.....	-----	7,700	-----	-----	-----	18	12	42	10	1	17	239,000
June 14.....	4,260,000	8,200	-----	-----	-----	43	18	25	-----	-----	14	260,000
June 15.....	4,350,000	13,550	-----	-----	-----	20	39	23	4	1	13	261,000
June 17.....	4,350,000	10,750	85	-----	-----	14	54	26	3	-----	3	324,000

He was given transfusions of 300 cc. of blood daily for a period of 5 days. The blood picture returned to normal and the patient was considered completely recovered in 22 days from onset of symptoms.

MAPHARSEN—(47-1939).—After exposure to infection on December 22, 1938, this patient developed a penile lesion which was positive for *Treponema pallidum*, A Kahn blood test was 4 plus.

From February 12 to July 3, 1939, he received 16 injections of neoarsphenamine. Twelve hours after the last injection of neoarsphenamine an arsenical dermatitis developed consisting of a macular rash on the dorsum of the hands and feet and antecubital areas. Recovery from this reaction was considered complete in 9 days. A discussion of this reaction will be found in the October 1940 number of the Naval Medical Bulletin, p. 589, case (2-1939).

Following the above reaction weekly injections of bismuth subsalicylate were administered. After the third injection a white blood count was 9,500; polys. 55; lymphs. 29; eosins. 4; immatures, 2. The patient complained of constipation and feeling below par. About 7 weeks after the reaction from neoarsphenamine, arsenical treatment was resumed and 0.06-gram injections of mapharsen were administered on August 22, 29, September 5, 12, and 20.

Blood picture

Date	WBC.	Polys.	Lymphs.	Eosins.	Imma- ture	Bands	Trans.	Basos.
1939								
September 21.....	9,700	55	29	13	2	-----	1	-----
September 25.....	15,050	49	25	22	-----	2	-----	2
September 26.....	15,060	49	25	22	-----	-----	-----	2
October 3.....	15,800	53	20	22	1	4	-----	1
October 10.....	7,750	69	21	8	-----	2	-----	-----
October 16.....	9,860	52	39	5	4	2	-----	-----
October 24.....	9,600	66	18	12	1	2	-----	1
October 30.....	9,600	67	23	4	4	2	-----	-----

Nineteen 1-gram injections of sodium thiosulphate were administered intravenously. Recovery in 39 days from onset of first symptoms.

FATAL REACTIONS

(48-1939).—A patient, exposed to infection December 16, 1938, was given a diagnosis of syphilis because of a positive darkfield examination and a positive Kahn blood test.

Antiluetic treatment was instituted with a 0.3-gram injection of neoarsphenamine on December 21, 1938, followed by a 0.45-gram injection on December 24 and 0.6-gram injections on December 28, and January 4 and 10, 1939. Three injections of bismuth subsalicylate were given as concurrent treatment.

Six hours after the last injection of neoarsphenamine the patient complained of severe sore throat and general malaise. Examination revealed an acutely inflamed throat with exudate on the tonsils. Temperature 103° F., pulse 120, respirations 24.

January 11: Diagnosis changed to tonsillitis, acute (intercurrent).

January 13: Dysphagia. Neck glands markedly swollen. WBC. 4,850; monos. 76; segmented 1; lymphs. 23; suggestion of infectious mononucleosis in smear.

January 14: Temperature septic in type, reading as high as 104° F. at times. Treatment with intravenous fluids. Ten cubic centimeters of pentnucleotide administered this date.

January 16: Diagnosis changed to infectious mononucleosis. Patient states he feels better. Temperature septic in character, ranging from 101° to 104° F. Less difficulty experienced in taking fluids by mouth, but the patient is still having difficulty in swallowing. There is a generalized macular tenderness. Several superficial veins in both arms are thrombosed.

January 17: General condition worse and the patient is less rational. Unable to take fluids, nasal tube passed. Pain over left lower chest. Large amounts of mucous bubbling rales heard over left base anteriorly and posteriorly. Pulse 110; respirations 20 and not labored. Heart sounds of poor quality and give the impression of gallop rhythm. Pentnucleotide 20 cc. daily in an effort to stimulate granulocyte production. The diagnosis of agranulocytosis following the administration of neoarsphenamine considered in view of patient's general course, blood picture, and character of his throat condition. Secondary bronchopneumonia suspected.

January 18: Laboratory reports and clinical course are more suggestive of an agranulocytosis than of an infectious mononucleosis. Diagnosis changed to granulocytopenia, malignant. There is a history of treatment of acute gonorrhea with large doses of sulfanilamide (100 grains per day) over an unknown period of time between November 10 and December 10, 1938. The patient was admitted to the hospital on December 19, a period of 9 days after last possible date of any medication. It is probable that the present condition has resulted from an arsenical poisoning with neoarsphenamine rather than the result of sulfanilamide therapy. The patient is critically ill. The respiration is 52 and quite labored with dilating ala nasi. He is somewhat stuporous and is aroused with difficulty. The throat is covered with white necrotic membrane. He is spitting up large amounts of mucopurulent material. Mucous bubbling rales in left base anteriorly and posteriorly. Pulse 114. Heart sounds of poor quality and still give the impression of gallop rhythm. Smear for K. L. B. negative. Temperature, 101.2° F.; pulse, 120; respirations 60 and quite labored. Patient placed in oxygen tent. Respiration ceased suddenly and patient was pronounced dead at 2:40 p. m., January 18, 1939, 8 days after onset of first symptoms.

Blood picture

Date	RBC	WBC	Hgb.	Bands.	Segs.	Lymphs.	Monos.	Eosins.
1939								
January 12.....	5,020,000	4,850	87			23	76	
January 14.....		6,500		1		35	64	
January 15.....		8,250				10	90	
January 16.....	5,200,000	7,500	81		2	15	83	
January 17.....		8,300		2	4	17	77	
January 18.....	5,000,000	3,500	81			12	86	2

(49-1939).—After exposure to infection on October 20, 1938, this patient (Pensioner, U. S. Navy) developed a penile lesion. Darkfield examinations were negative; 4-plus Kahn blood tests; a smear of urethral discharge was positive for gonococci.

A 0.45-gram injection of neoarsphenamine was administered on November 29, 1938, followed by 0.6-gram injections on December 6, 13, 20, 27, 1938, and January 3, 10, 17, and 24, 1939, a total of 5.25 grams. Daily mercury inunctions were given as concurrent treatment.

A 10-day course of sulfanilamide (420 grains) was begun on December 10, 1938. The patient tolerated all treatment without side reactions.

On January 26, 1939, two days after the ninth and last injection of neoarsphenamine, the patient complained of pain in the chest, spitting up of blood, and a slight sore throat. No evidence of acute pulmonary disease was found. X-ray of chest was negative. Temperature, 99.2° F.; pulse, 80; respirations, 20; WBC.; 1,700; RBC., 3,280,000; hgb., 62; bands., 3; segmented., 8; lymphs., 83; monos. 8.

During the period from January 26 to February 16 the red-blood count was approximately 3,500,000 or higher; the white count fluctuated between 1,200 and 1,700; the lymphocytes remained fairly stable at 96 percent, except for the first day when they were 83 percent; and the platelet count gradually decreased from 74,000 on January 29 to 3,000 on February 16.

A diagnosis of granulocytopenia, malignant, was made on February 14. The treatment for this condition was as follows: (a) Leukocytic extract, 5 cc. intragluteally, six times daily for first 5 days; (b) pentnucleotide, 10 cc. intragluteally, four times daily throughout course of the disease from the second day; (c) blood transfusions, both fractional and full when indicated, for a total of 2,650 cc.; (d) liver extract, two capsules orally, three times a day; and (e) vitamins B and C during last 10 days of disease.

In spite of active supportive treatment, the patient's general condition became progressively worse as manifested by weakness, nervousness, and loss of morale. There was constant bleeding from the right nostril during the last 10 days of illness. On February 16, 1939, the patient died, 21 days after onset of symptoms.

(50-1939).—After exposure to infection on September 2, 1938, this patient developed a penile lesion. A darkfield examination was positive for *Treponema pallidum*.

From October 11, 1938, to February 21, 1939, he received 16 injections of neoarsphenamine, a total of 7.4 grams, and 10 intramuscular injections of bismuth salicylate as concurrent treatment. Two days after the last injection of neoarsphenamine the patient complained of vague distress in the upper abdomen and lack of appetite. Physical examination revealed no other symptoms.

On February 27 a 0.03-gram injection of mapharsen was administered, and 2 days later the patient reported to the dispensary late in the day stating that he had been nauseated while on watch. He was admitted and treated symptomatically.

March 2: Examination showed patient to be distinctly jaundiced. Icterus index 16. WBC. 8,900 with differential count within normal limits. Liver enlarged; spleen enlarged to percussion.

March 4: The patient was nauseated but able to retain water and orange juice. A total of 1,000 cc. of 5-percent dextrose in saline solution was given intravenously.

March 6: Patient delirious and unable to retain anything by mouth. Icterus index, 100; blood bilirubin, 13.2 mgs. and 17 mgs. Urine and stool positive for urobilin. Urine showed a trace of albumin and sugar.

March 7: Late in the afternoon of this date, coma deepened, respirations became deep, labored, and rapid. Oxygen given. Pulmonary edema developed with coarse bronchi noted over entire chest. Respirations became weaker, and on March 7 patient died, 6 days from onset of symptoms.

Autopsy findings.—Acute necrosis of liver; jaundice; petechial hemorrhage in skin, serous surfaces, lungs, and gastro-intestinal tract; acute splenic tumor; peritoneal effusion; ascites; edema of intestines; purulent bronchitis; early bronchopneumonia; fresh rheumatic endocarditis of aortic valve; small scars in lungs; healed chancre of penis.

STATISTICS

HEALTH OF THE NAVY

The statistics (annual rates per 1,000) appearing in this summary were compiled from data contained in monthly reports of communicable diseases received in the Bureau for the months of April, May, and June 1940:

ENTIRE NAVY

Year	All diseases	Injuries and poisonings	All causes	Communicable diseases		Venereal diseases
				A	B	
1935.....	371	67	438	28	85	62
1936.....	337	49	386	30	140	42
1937.....	276	36	313	18	98	59
1938.....	331	50	382	9	79	78
1939.....	339	48	387	7	85	90
1940.....	431	51	482	48	118	83

FORCES ASHORE

1935.....	491	80	571	54	110	45
1936.....	518	50	568	59	226	26
1937.....	312	36	347	34	131	27
1938.....	364	51	415	15	105	45
1939.....	332	48	380	12	104	41
1940.....	446	53	498	43	144	52

FORCES AFLOAT

1935.....	312	60	372	16	72	71
1936.....	229	49	278	13	89	51
1937.....	256	37	293	8	78	78
1938.....	313	50	363	5	64	96
1939.....	343	47	391	4	75	117
1940.....	420	50	470	52	99	105

Common infectious diseases of the respiratory type.—These diseases caused 6,752 admissions during April, May, and June, 1940, or a 114 percent increase when compared with the corresponding quarter in 1939. Catarrhal fever was responsible for 3,763 of the total admissions for respiratory diseases. There were 3,985 admissions reported by forces afloat, 2,713 from shore stations in the United States, and 54 from outlying naval stations and activities. Ships and shore

stations reporting the greatest number of admissions for the quarter were as follows:

Ship or station	April	May	June	Total
Naval training station, Norfolk, Va.....	199	219	136	554
Naval training station, Newport, R. I.....	234	183	69	486
Naval training station, Great Lakes, Ill.....	189	0	99	288
Marine Barracks, Quantico, Va.....	73	49	39	161
U. S. S. <i>Saratoga</i>	64	58	25	147
Marine Corps Base, San Diego, Calif.....	55	39	47	141
U. S. S. <i>Vestal</i>	23	85	14	122
Naval air station, Pensacola, Fla.....	27	33	35	95
Naval training station, San Diego, Calif.....	43	37	15	95
U. S. S. <i>Enterprise</i>	39	28	24	91
U. S. S. <i>Helena</i>	7	35	30	72
U. S. S. <i>Arizona</i>	18	39	11	68
U. S. S. <i>Augusta</i>	8	25	29	62
U. S. S. <i>Wasp</i>	2	40	19	61
U. S. S. <i>Maryland</i>	19	28	13	60
U. S. S. <i>Tennessee</i>	21	12	19	52
Receiving ship, New York, New York.....	36	9	7	52
U. S. S. <i>Idaho</i>	24	13	14	51
U. S. S. <i>New Mexico</i>	15	17	19	51
U. S. S. <i>Savannah</i>	10	19	21	50
U. S. S. <i>Mississippi</i>	13	22	11	46
U. S. S. <i>West Virginia</i>	17	14	13	44
Naval air station, Norfolk, Va.....	21	18	5	44
U. S. S. <i>California</i>	16	14	13	43
Submarine base, New London, Conn.....	16	6	16	38
Naval Academy (Midshipmen).....	31	6	1	38
Submarine base, Coco Solo, C. Z.....	15	12	10	37
U. S. S. <i>Honolulu</i>	13	15	5	33

On May 16, 1940, an epidemic of tonsillitis, acute, appeared on the U. S. S. *Vestal* and resulted in 74 admissions to the sick list. The causative organism was a hemolytic streptococcus. A carrier suspect was found, and the main scuttlebutt established as the source of dissemination. Suitable control measures brought the situation under control in 4 days.

The medical officer of a tender under date of July 12, 1940, reported an outbreak of 41 cases of acute pharyngitis, beginning on June 24, 1940. Cases, reported by divisions, were as follows:

First, 14; second, 3; third, 6; fourth, 4; fifth, not aboard; sixth, 1; seventh, 2; eighth, 4; ninth, 4; tenth (medical), 0; and officers, 3. There were 14 food handlers, 7 mess cooks, 3 mess attendants, 1 ship's cook, and 1 ship's baker among the patients. One man who had charge of ice box from which meats were issued and 1 fountain operator who worked for ship's service were also admitted to the sick list.

All cases showed injection of the pharynx and soft palate and complained of sore throat; 10 cases showed signs of rhinitis, 12 cases had a cough, 3 cases complained of general malaise, and 4 cases showed elevation of temperature (maximum, 101° F.).

It is the opinion of the reporting medical officer that this outbreak was due to overcrowding, a total of 645 men being aboard a vessel designed to accommodate 434.

Other infectious diseases.—A total of 797 cases of german measles and 156 cases of measles occurred during the quarter, the largest number of cases being reported from the following ships and stations:

Ship or station	German measles	Measles
U. S. S. <i>Argonne</i>	34	0
U. S. S. <i>Arizona</i>	36	0
U. S. S. <i>Arkansas</i>	6	33
U. S. S. <i>California</i>	47	4
U. S. S. <i>Chaumont</i>	20	0
U. S. S. <i>Henderson</i>	56	0
U. S. S. <i>Maryland</i>	23	6
U. S. S. <i>New Mexico</i>	40	0
U. S. S. <i>New York</i>	54	10
U. S. S. <i>Oklahoma</i>	26	0
U. S. S. <i>Ranger</i>	62	0
U. S. S. <i>Richmond</i>	45	0
U. S. S. <i>Saratoga</i>	21	0
U. S. S. <i>Tennessee</i>	41	15
U. S. S. <i>Texas</i>	2	26
U. S. S. <i>West Virginia</i>	19	2
U. S. S. <i>Wichita</i>	29	5
U. S. S. <i>Wyoming</i>	52	1
U. S. S. <i>Yorktown</i>	42	1
Naval training station, Norfolk, Va.....	238	20
Naval training station, San Diego, Cal.....	80	8
3d Defense battalion, Pearl Harbor, T. H.....	34	0

A total of 241 cases of mumps was reported for the second quarter of 1940. The Marine detachment, Peiping, China, reported 16 cases in April and 7 cases in May; the Naval training station, Great Lakes, Ill., 4 cases in April, 32 in May, and 22 in June; the U. S. S. *Maryland*, 6 cases in April, 12 in May, and 19 in June; the U. S. S. *Mississippi*, 3 cases in April, 14 in May, and 4 in June; and the U. S. S. *Tennessee*, 1 case in April, 13 in May, and 8 in June.

Eighteen cases of chickenpox were reported for the quarter, 9 from forces afloat and 9 from forces ashore.

Single cases of scarlet fever occurred in April on board the U. S. S. *Sonoma* and at the receiving ship, New York, New York, and the naval powder factory, Indian Head, Maryland; and in June at the naval training station, Great Lakes, Illinois.

The Fourth Marines, Shanghai, China, reported 1 case of poliomyelitis in June.

An apprentice seaman, U. S. N. R., 17 years of age, with 7 months' service, was admitted to the sick list with cerebrospinal fever, meningococcic, on board the U. S. S. *Henderson* on May 1, 1940, and transferred to the regimental hospital, Shanghai, China. He was returned to duty on May 15, 1940.

A seaman, first class, 21 years of age, with 3 years' service, was admitted with cerebrospinal fever, meningococcic, on board the U. S. S. *Maryland* on April 26, 1940, and transferred to the U. S. S. *Relief* the same day. He was returned to duty after 28 days on the sick list.

A private, Marine Corps, 20 years of age, with 1 year and 5 months' service, on duty with the Fourth Marines, Shanghai, China, was transferred to regimental hospital on June 15, 1940, with meningitis, cerebrospinal, acute. The patient developed edema of the lungs and died on June 19.

A signalman, first class, 26 years of age, with 8 years and 3 months' service, was admitted with paratyphoid fever on board the U. S. S. *Pecos* on April 29, 1940, and discharged to duty on June 7. The questionnaire for this case has not been received in the Bureau, consequently no information is available concerning type, severity of case, and history of prophylaxis.

A moderately severe, uncomplicated case of typhus fever was admitted on board the U. S. S. *Henderson*, May 12, 1940, and transferred to the regimental hospital, Shanghai, China, May 19. The patient was a sergeant, Marine Corps, 32 years of age, with 7 years and 7 months' service. He was discharged to duty June 14.

Venereal disease.—The following table of statistical data indicates the frequency of occurrence of venereal diseases during April, May, and June 1940, as compared with the 5-year median for the corresponding quarter:

Ship	Admission rate per 1,000	Median rate, 2d quarter, 1935-39
Arkansas.....	100. 90	63.76.
Boise.....	152. 52	Commissioned Aug. 12, 1938.
Chester.....	60. 70	41.18.
Enterprise.....	178. 96	Commissioned May 12, 1938.
Helena.....	167. 12	Commissioned Sept. 18, 1939.
Langley.....	551. 15	55.17.
Memphis.....	141. 37	112.56.
Nashville.....	103. 76	Commissioned June 6, 1938.
Omaha.....	236. 22	159.42.
Ranger.....	194. 59	55.58.
St. Louis.....	176. 62	Commissioned May 19, 1939.
Tennessee.....	104. 43	75.97.
Texas.....	100. 54	108.08.
Trenton.....	191. 45	102.56.
Tuscaloosa.....	114. 30	63.31.
Vincennes.....	117. 99	Commissioned Feb. 24, 1937.
Wichita.....	110. 28	Commissioned Feb. 16, 1939.
Wyoming.....	125. 44	198.17.

Gastro-enteritis, acute.—The medical officer of a tender, under date of August 31, 1940, reported that 14 cases of acute gastro-enteritis were admitted to the sick list and 13 additional cases received treatment for mild symptoms of short duration from June 4 to June 8.

This vessel departed from San Diego, Calif., on June 1 and arrived at Pearl Harbor, T. H., on June 9.

Twenty-seven cases were admitted to the sick list from June 18 to June 30, and 32 cases were admitted in July. The average sick days per case was 2.6 for June and 3.7 for July. The infection appears to have been introduced by a case of bacillary dysentery contracted prior to leaving the States or by a carrier among the crew or passengers.

The onset was sudden and characterized by abdominal cramps, low-grade fever, and diarrhea. The course was mild in the majority of cases, symptoms subsiding in from 12 to 24 hours. Several patients gave a history of headache, muscular soreness, neuritic pains, and chilly sensations 6 to 8 hours prior to the onset of the abdominal symptoms. In these cases the diarrhea and a fever of 102° F. to 103° F. persisted for 48 to 72 hours.

Physical findings were not significant except for moderate abdominal tenderness. Microscopical fecal examinations were negative for ova and parasites. Red blood cells and mucus were present in the stools of the moderately severe cases. Fecal cultures made on the U. S. S. *Relief* and at the United States Naval Hospital, Pearl Harbor, T. H., were positive for *Shigella paradysenteriae* (Hiss).

Treatment was symptomatic and consisted of bed rest, bland diet, forced fluids, bismuth subnitrate and opiates. Strict isolation of cases was a difficult procedure, especially for the 5 cases occurring among the officer personnel.

Water received by barge from the navy yard, Pearl Harbor, and evaporated water was used for all purposes, except for the fire mains and flushing systems. Culture of the ship's water supply was negative for colon bacilli. Fresh fruits, vegetables, and milk obtained locally were apparently free from contamination as other ships using the local products were free from gastro-intestinal disorder.

After investigation the battle force medical officer concluded that the outbreak was occasioned by the dysentery organism *Shigella paradysenteriae* (Hiss) and that the spread on board ship was effected through random direct and indirect passage from person to person. The force medical officer stated that the inadequate toilet facilities for the enlisted men must be considered a potential source of danger to their health, particularly when the ratio of toilets to enlisted men on this vessel is 1 to 38, as compared with the standard of 1 to 18.

Food poisoning.—(1) An outbreak of food poisoning occurred May 10, 1940, among the recruits attached to the outgoing unit at a naval training station. A total of 123 men were admitted to the hospital. Possibly 50 to 75 others suffered slight attacks but did not report to the sick bay for treatment.

There was marked and sudden prostration and diarrhea. Nausea and vomiting continued for about 6 to 8 hours. Continuous epi-

gastric pains and frontal headaches were reported in a number of cases. No ocular symptoms were observed. The suspected food was creamed eggs on toast served at breakfast on May 10. Onset occurred about 4 hours after breakfast and seemed to affect men who ate at 0715, or during the latter part of the breakfast serving, which would indicate that the food was deteriorating rapidly. This food was eaten by each man affected, but others in this same group ate the eggs and showed no symptoms. Only 4 of the 80 men served at the beginning of the meal were stricken.

No food was available for examination. Specimens of the vomitus and stool in the cases of 6 of the most severely ill were put in beef extract bouillon and showed almost a pure culture of *Staphylococcus albus*. On Endo's agar the stool specimens showed many *Escherichia coli* and some communioid and on plain agar a few colonies of *Staphylococcus albus*. The specimens were negative for *Salmonella* and *Shigella* groups.

Most of the 123 men transferred to hospital had recovered the following day.

(2) An outbreak of 12 cases of food poisoning occurred on a destroyer on May 3, 1940. The suspected food was ice cream served at the noon meal. It was mixed at 6 p. m. the previous day, set aside in a warm galley, and placed in the refrigerator at 8:30 p. m. The chief commissary steward saw no evidence of spoilage of any item used in the ice cream. The mixture was removed from the refrigerator the following morning, at which time the cook noted a slight curdy appearance in two of the three containers. A slight bitter taste in the ice cream was noted by several members of the crew at time of ingestion. Three chief petty officers became ill within 30 minutes after eating an additional portion of the ice cream at 2:30 p. m. No other chief petty officers were affected although all had eaten the ice cream at the noon meal. The ice cream was the only food eaten by all cases. It was eaten by a large number who were not affected.

Identical clinical manifestations were noted in the 12 cases admitted to the sick list: Prostration, nausea, vomiting, abdominal cramps, diarrhea, profuse perspiration, dilated pupils but no visual disturbance, chills, and shock. Moderately severe leg cramps were noted in three cases. Onset was within 2 to 4 hours after ingestion of noon meal. Treatment was eliminative, symptomatic, and supportive and all cases were sufficiently recovered to carry on with their regular duties the following day.

No item of food was available for examination.

Conclusions: (a) Ice cream was the offending food; (b) the condensed milk probably contained the specific toxic substance; (c) the time element between mixing of formula and refrigeration was an im-

portant contributory factor in development of the toxic agent; and (d) closer supervision of food preparation is indicated.

(3) A destroyer reported 10 cases of food poisoning admitted to the sick list on June 11 while the vessel was completing the regular overhaul period at a navy yard. Twelve additional men were ill but remained on light duty and 6 others recalled a slight diarrhea.

The suspected food was potato salad which was allowed to remain in the galley from 1300 when it was prepared until 1700 when it was served. The temperature and humidity of the galley were unusually high during the period. No other single item of food was eaten by all who became ill.

There was sudden onset of headache and nausea without previous disease or symptoms. Spasmodic abdominal pain, profuse diarrhea, vomiting, chills and fever, rapid and regular pulse rates, and normal respiration, were noted in all cases. Frontal and occipital headaches occurred. Hematemesis was reported in 1 case. Symptoms appeared in some cases within 8 hours, but in most cases developed within 12 to 16 hours.

Blood counts and urine were normal in all respects. Examinations of the stools were negative for ova or parasites. Cultures were not taken. None of the potato salad remained for examination.

Compressed-air illness.—Five cases of mild compressed-air illness were reported by the medical officer of the naval torpedo station, Newport, R. I., under date of July 15, 1940.

Case No.	Rate	Depth of dive	Time on bottom (min.)	Decompression (feet)	Symptoms	Recompressed (feet)	Decompressed (time)
1	SF-1C-----	123	42	120-132	Severe rash; itching.	150	5 hrs. 14 min.
2	TM-1C-----	126	48	120-132	In right arm and left leg.	150	3 hrs. 5 min.
3	SF-1C-----	116	81	108-120	In left arm-----	^a 128	3 hrs. 10 min.
4	CBM-(master diver).	122	59	120-132	-----do-----	180	1 hr. 51 min.
5	CBM-----	152	71	144-156	In right shoulder. Weakness in left leg.	200	(^b)

^a Inability to equalize pressure caused pain in ears which prevented further descent.

^b Oxygen used after reaching 60 feet.

The medical officer was of the opinion that these cases of compressed-air illness were caused by frequent dives requiring excessive physical exertion against strong tides and with large line areas.

MORBIDITY

Summary for the quarter ending June 30, 1940

Average strength.....	Forces afloat 105,561		Forces ashore 75,984		Entire Navy 181,545	
	Admis- sions	Rate per 1,000	Admis- sions	Rate per 1,000	Admis- sions	Rate per 1,000
All causes.....	12,408	470.17	9,461	498.05	21,869	481.84
Diseases only.....	11,081	419.89	8,463	445.51	19,544	430.61
Injuries and poisonings.....	1,327	50.28	998	52.54	2,325	51.23
Communicable diseases trans- missible by oral and nasal discharges (class VIII):						
(A).....	1,371	51.95	816	42.96	2,187	48.19
(B).....	2,602	98.60	2,737	144.08	5,339	117.63
Venereal diseases.....	2,764	104.74	981	51.64	3,745	82.51

DEATHS

During the quarter ending June 30, 1940

Cause		Navy			Marine Corps		Nurse Corps	Total
Principal	Secondary or contributory	Offi- cers	Mid- ship- men	Men	Offi- cers	Men		
Average strength.....		12,544	2,064	139,499	1,697	25,299	442	181,545
<i>Disease</i>								
Adenocarcinoma, sigmoid	Peritonitis, general, acute	1						1
Alcoholism, acute	None			1				1
Appendicitis, acute	Peritonitis, general, acute	1		3				4
Cerebrospinal fever, men- ingococcic	Abscess, brain			1				1
Embolism, cerebral	None			1				1
Endocarditis, acute	do			1				1
Hemorrhage, cerebral	do			1				1
Do	Arteriosclerosis, general					1		1
Do	Hypertension, arterial	1						1
Hemorrhage, subarach- noid	do			1				1
Hypernephroma	None			1				1
Leukemia, chronic, mye- logenous	Absence, acquired, spleen			1				1
Malaria, malignant ter- tian	None			1				1
Meningitis, cerebrospinal, acute	Edema, lung					1		1
Pneumonia, broncho	None					1		1
Do	Endocarditis, acute			1				1
Syphilis	Thrombosis, coronary, artery			1				1
Thrombosis, cerebral	Arteriosclerosis, general	1						1
Thrombosis, coronary, ar- tery	None	3						3
Do	Coronary heart disease, ar- teriosclerotic			1				1
Tuberculosis, general mil- itary	None			1				1
Do	Meningitis, cerebrospinal, acute			1				1
Tuberculosis, pulmonary, chronic, active	None					1		1
Tuberculosis, genito-uri- nary tract	do			1				1
Total for diseases.....		7	0	18	0	4	0	29

Deaths during the quarter ending June 30, 1940—Continued

Cause		Navy			Marine Corps		Nurse Corps	Total
Principal	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
<i>Injuries and poisonings</i>								
Asphyxiation.....	None.....			2				2
Castration, traumatic.....	Not determined.....			1				1
Drowning.....	None.....			2		2		4
Do.....	Alcoholism, acute.....			2				2
Do.....	Intracranial injury.....			1				1
Do.....	Rupture, traumatic, liver.....			1				1
Electric shock.....	None.....			1				1
Fracture, compound, skull.....	do.....	1		1		2		4
Fracture, simple, skull.....	Abscess, brain.....			1				1
Do.....	Intracranial injury.....			1		1		2
Fracture, simple, vertebra, cervical.....	Intraspinal injury.....					1		1
Injuries, multiple, extreme.....	None.....	4		7		1		12
Do.....	Peritonitis, general, acute.....			1				1
Intracranial injury.....	None.....			4				4
Strangulation, respiratory.....	do.....			1				1
Wound, gunshot:								
Abdomen.....	do.....			1				1
Chest.....	do.....			1				1
Head.....	do.....					1		1
Heart.....	do.....			1				1
Poisoning, acute:								
Cyanide.....	do.....			1				1
Phenol.....	do.....			1				1
Sodium fluoride.....	do.....			1				1
Total for injuries and poisonings.....		5	0	32	0	8	0	45
Grand total.....		12	0	50	0	12	0	74
Annual death rate per 1,000:								
All causes.....		0.96		0.36		0.47		0.41
Diseases only.....		.56		.13		.16		.16
Drowning.....				.04		.08		.04
Poisoning.....				.02				.02
Other injuries.....		.40		.16		.24		.19

MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

Statistics for the quarter ending June 30, 1940

The following statistics were taken from sanitary reports submitted by naval training stations.

April, May, and June 1940	Naval training station			
	Norfolk, Va.	Newport, R. I.	Great Lakes, Ill.	San Diego, Calif.
Recruits received during the period.....	2, 926	1, 923	2, 643	2, 818
Recruits appearing before Board of Medical Survey.....	43	34	22	0
Recruits recommended for discharge from the Service.....	38	27	22	23
Recruits discharged by reason of Medical Survey.....	25	(a)	14	(a)
Recruits held over pending further observation.....	20	15	(a)	0
Recruits transferred to the hospital for treatment, operation, or further observation for conditions existing prior to enlistment.....	0	24	(a)	49

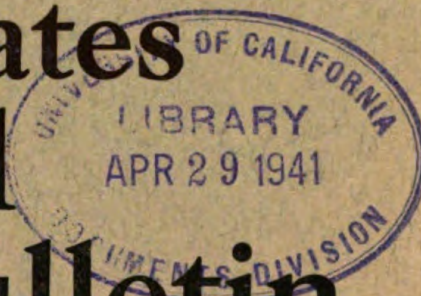
(a) Not reported.

The following table was prepared from reports of medical surveys in which disabilities or disease causing the survey were noted existing

prior to enlistment. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office.

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Absence, acquired, toe	1	Loose body in joint	
Absence, acquired, teeth	2	Malocclusion, teeth	5
Abscess, periapical	1	Mental deficiency, moron	2
Acne	1	Migraine	1
Amblyopia	2	Metatarsalgia	1
Amebiasis	1	Myopia	2
Amputation, traumatic	1	Myositis, chronic	1
Ankylosis, joint	1	Nephritis, chronic	13
Arthritis, chronic	2	Neuritis, sciatic nerve	1
Asthma	5	Neuroma	1
Astigmatism	4	Nostalgia	1
Cardiac arrhythmia, paroxysmal tachycardia	1	Obstruction, intestinal, from spastic or paralytic causes	1
Cardiac arrhythmia, premature contraction	1	Osgood-Schlatter disease	3
Caries, teeth	4	Osteochondritis deformans	1
Cicatrix, skin	1	Osteochondritis dissecans	1
Constitutional psychopathic inferiority, without psychosis	3	Otitis media, chronic	16
Constitutional psychopathic state, emotional instability	1	Pansinusitis	1
Constitutional psychopathic state, inadequate personality	6	Paradentosis	5
Curvature, spine	1	Perforated nasal septum	1
Cyst, teratoma, quiescent	1	Periostitis, chronic	1
Deafness, bilateral	1	Pes cavus	2
Deafness, unilateral	2	Pneumonitis, chronic, nontuberculous	1
Deformity, acquired	11	Psoriasis	1
Deformity, congenital	9	Prolapse, rectum	1
Dementia praecox	8	Prostatitis, chronic	1
Dermatitis, seborrheica	1	Psychoneurosis, hysteria	1
Diabetes mellitus	2	Psychoneurosis, neurasthenia	2
Dislocation, articular cartilage	4	Psychoneurosis, psychasthenia	2
Enuresis	21	Psychosis, intoxication, alcoholic	1
Encephalitis, chronic	1	Psychosis, manic-depressive	1
Epilepsy	16	Rheumatic fever	3
Epilepsy, Jacksonian	1	Sinusitis, maxillary	1
Flat foot	26	Somnambulism	6
Fungus infection, skin	1	Syncope	2
Gastritis, chronic	1	Synovitis, chronic	1
Genu recurvatum	1	Syphilis	1
Glycosuria	2	Splanchnoptosis	1
Gonococcus infection, urethra	7	Sprain, joint	1
Gonococcus infection, prostate	1	Trachoma	1
Headache	1	Tuberculosis, pulmonary, chronic, active, moderately advanced	1
Hernia, epigastric	1	Tuberculosis, urogenital	1
Hernia, inguinal, direct	1	Ulcer, duodenum	2
Hernia, inguinal, indirect	7	Ulcer, stomach	1
Hyperopia	1	Union of fracture, faulty	1
Hypertension, arterial	2	Valvular heart disease, mitral stenosis	2
Hypothyroidism	1	Varicocele	1
Intraspinal injury	1		
Jaundice, hemolytic, familial	1	Total	271
Joint, internal derangement of	1		

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UNITED STATES NAVAL MEDICAL BULLETIN

FOR THE INFORMATION OF
THE MEDICAL DEPARTMENT OF THE NAVY



DIVISION OF PUBLICATIONS
THE BUREAU OF MEDICINE AND SURGERY



THE MISSION OF THE MEDICAL DEPARTMENT OF THE NAVY



TO KEEP AS MANY MEN AT AS MANY GUNS AS
MANY DAYS AS POSSIBLE



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NAVY DEPARTMENT,
Washington, March 20, 1907.

This UNITED STATES NAVAL MEDICAL BULLETIN is published by direction of the Department for the timely information of the Medical and Hospital Corps of the Navy.

TRUMAN H. NEWBERRY,
Acting Secretary.

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PREFACE

THE UNITED STATES NAVAL MEDICAL BULLETIN was first issued in April 1907 as a means for supplying medical officers of the United States Navy with information regarding the advances which are continually being made in the medical sciences, and as a medium for the publication of accounts of special researches, observations, or experiences of individual medical officers.

It is the aim of the Bureau of Medicine and Surgery to furnish in each issue special articles relating to naval medicine, descriptions of suggested devices, clinical notes on interesting cases, editorial comment on current medical literature of special professional interest to Medical Department personnel, and reports from various sources, notes, and comments on topics of professional interest.

The Bureau extends an invitation to all medical and dental officers to prepare and forward, with a view to publication, contributions on subjects of professional interest.

In order that each service contributor may receive due credit for his efforts in preparing matter for the BULLETIN of distinct originality and special merit, the Surgeon General of the Navy will send a letter of appreciation to authors of papers of outstanding merit.

The Bureau does not necessarily undertake to endorse views or opinions which may be expressed in the pages of this publication.

ROSS T. McINTIRE,
Surgeon General, United States Navy.

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NOTICE TO CONTRIBUTORS

Contributions to the BULLETIN should be typewritten, double spaced, on plain paper, and should have wide margins. Fasteners which will not tear the paper when removed should be used. Nothing should be written in the manuscript which is not intended for publication. For example, addresses, dates, etc., not a part of the article, require deletion by the editor. The BULLETIN endeavors to follow a uniform style in heading and captions, and the editor can be spared much time and trouble, and unnecessary changes in manuscript can be obviated if authors will follow in these particulars the practice of recent issues.

The greatest accuracy and fullness should be employed in all citations, as it has sometimes been necessary to decline articles otherwise desirable because it was impossible for the editor to understand or verify references, quotations, etc. The frequency of gross errors in orthography in many contributions is conclusive evidence that authors often fail to read over their manuscripts after they have been typewritten.

Contributions must be received at least 3 months prior to the date of the issue for which they are intended.

The editor is not responsible for the safe return of manuscripts and pictures. All material supplied for illustrations, if not original, should be accompanied by reference to the source and a statement as to whether or not reproduction has been authorized.

The BULLETIN intends to print only original articles, translations, in whole or in part, reviews, and reports and notices of Government or departmental activities, official announcements, etc. All original contributions are accepted on the assumption that they have not appeared previously and are not to be reprinted elsewhere without an understanding to that effect and that editorial privilege is granted to this Bureau in preparing all material submitted for publication.

EBEN E. SMITH, *Editor.*

Commander, Medical Corps, United States Navy.

SPECIAL ARTICLES

PHYSIOLOGICAL EFFECTS OF HIGH ALTITUDE¹

By Lieutenant A. E. Behnke, Medical Corps, United States Navy, and Lieutenant T. L. Willmon, Medical Corps, United States Navy

In consideration of the physiological responses induced by changes in atmospheric pressure incident to high-altitude flying, it is necessary to distinguish between primary pressure phenomena and the reactions resulting from alterations in gas pressures in the lungs and within the body tissues.

PRIMARY PRESSURE PHENOMENA

THE PARANASAL SINUSES AND EAR: Apart from the disturbances in gaseous equilibria in tissues, pressure variations in the range of $\frac{1}{2}$ atmosphere to 15 atmospheres are apparently without physiologic effect, provided that equalization of pressure takes place in the spaces of the ear and sinuses.

When the body is subjected to increased pressure, every air space from the smallest and most inaccessible ethmoid cell to the air cells in the tip of the mastoid process receives air, provided that the passages to these spaces are open (fig. 1). If a sinus opening or auditory tube is obstructed, as is illustrated in figure 1, an increased pressure of 1 to 2 pounds transmitted to the body tissues and circulating blood produces an area of relatively decreased pressure in the air spaces, thus effecting a cupping action in the cavity and a vascular congestion of the mucous membrane. Tension is produced on the cells of the lining membrane, and blood vessels are dilated in tissue surrounding the occluded space. Pain is present in and around the involved area, associated with congestion and hemorrhage. If an auditory tube is blocked, pain will be felt in the region of the tympanic membrane and frequently over the surface of the mastoid process. From sinus blockage, pain serves to outline the involved area on the surface of the head and face so that a diagnosis of maxillary, ethmoidal, or frontal sinus obstruction is evident. The important physiological principle is that differential pressure elicits pain.

CLINICAL OBSERVATIONS.—Acute or chronic upper respiratory tract infection, inducing occlusion of auditory tubes and paranasal sinus passages, is the most important factor preventing accommodation to

¹ From the laboratory of the Experimental Diving Unit, Navy Yard, Washington, D. C.

pressure variations. The prevalence of complete or partial blockage of one or both auditory tubes is shown by the fact that of 2,000 qualified submarine personnel, 10 to 15 percent were unable to tolerate pressure excess of about 2 pounds per square inch applied at the rate of 45 pounds per minute.

In similar tests on civilians a percentage of individuals as high as 50 may be unable to compensate for pressure changes.

The effects of variation in pressure, learned mainly from observations on deep-sea divers, apply equally well to aviation. In 1937 Armstrong and Heim² reported a clinical entity termed "aero-otitis

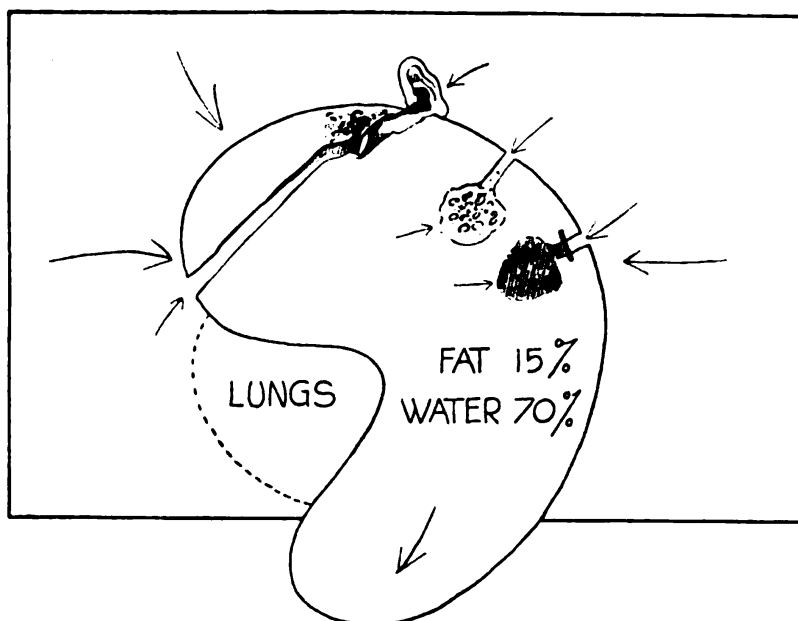


FIGURE 1.—Diagrammatic illustration of pressure effects on body spaces. (Published in *Annals of Internal Medicine*, June 1940. Reproduced by permission of the publishers.)

media," concerned with traumatic effects on the tympanic membrane and middle ear as a result of uncompensated pressure changes during altitude flight.

This entity is new only in its application to aviation, since traumatic pressure injury to the middle ear was observed about 100 years ago and carefully evaluated and described by Heller, Mager, and von Schrötter in 1900.³ Essentially, it consists of congestion and occasionally hemorrhagic extravasation in the tympanic membrane. The vessels over the handle of the malleus and inner portion of the auditory canal are usually dilated. The symptoms are pain of varying intensity, frequently tinnitus, and a temporary loss of auditory acuity. The etiology of the injury is a cupping action on the middle ear pro-

² Armstrong, H. G., and Heim, J. W.: Effect of flight on middle ear. *J. A. M. A.* 109: 417-421, August 1937.

³ Heller, E., Mager, W., and von Schrötter, H.: *Luftdruck-Erkrankungen*, vol. 1 and 2. A. Holder, Vienna, 1900.

duced by differences in pressure in the manner described in previous paragraphs.

Recently, however, we have observed a hitherto unreported entity consisting of congestion in the middle ear and appearing not immediately but 18 hours after prolonged inhalation of oxygen at high altitudes. The explanation for this phenomenon appears to be a negative pressure effect brought about by the absorption of oxygen from the middle ear spaces during sleep when voluntary opening of the auditory tubes is not effected.

Inhalation of air following removal of the oxygen mask at about the 10,000-foot level during altitude descent minimizes or abolishes the symptoms.

In a typical test run at simulated altitudes, oxygen is breathed during pressure decrease, during stay at the ceiling altitude, and during pressure increase corresponding to altitude descent. Under these conditions the air in the middle ear spaces including mastoid cells is flushed out and replaced by pure oxygen. Upon return to ground level, this oxygen is slowly absorbed by the tissues and circulating blood tending to produce a cupping action. Equalization of pressure in the ear spaces is effected voluntarily so that signs and symptoms of injury are not present.

During sleep, however, voluntary opening of the auditory tube is suppressed to create a condition of uncompensated negative pressure. Upon awakening in the morning the individual experiences sensations of fullness accompanied by pain and tenderness in the ears, tinnitus, and decreased hearing. Equalization of pressure in the middle ear spaces to relieve the symptoms requires the exertion of expiratory force which induces hissing and gurgling noises, and exacerbates the pain. Inspection of the ear drum now reveals intense redness with retraction, or occasional bulging simulating the appearance of acute otitis media. Auditory acuity may be reduced as much as 5/15 for whispered voice. About 6 hours later the symptoms and findings usually diminish without therapy and disappear in from 24 to 48 hours.

EFFECT OF PRESSURE VARIATIONS ON HEARING.—The problem of impaired hearing as a result of pressure changes has received considerable attention in aviation medicine. We have observed that pressure trauma to the tympanic membrane in varying degrees of severity, beginning with congestion and terminating in hemorrhage and perforation of the drum, is promptly repaired without permanent loss of auditory acuity.

Audiograms obtained on 19 deep sea divers subjected to pressure trauma over a period of 5 to 15 years show a diminution of hearing in the range of 4,096 double vibrations, beyond the range of the spoken voice and attributable to nerve injury as a result of noise. Audio-

grams made on 20 professional musicians showed similar patterns in comparable age groups.

The gas volume in the middle ear tends to be reduced by the force of compression exerted on the tympanic membrane and through the blood stream creating a condition normally compensated for by intermittent opening of the auditory tube.

In a descent from a simulated altitude of 30,000 feet to the ground level requiring 6 minutes, voluntary opening of the auditory tube occurred 18 times at relatively constant altitude decrements of 1,700 feet. The differential pressure, however, on the tympanic membrane when the auditory tube was voluntarily opened, gave values of 17, 24, 33, and 44 mm. of mercury corresponding to altitudes of 30,000, 20,000, 10,000, and 300 feet, respectively.

Likewise in a dive to a depth of 100 feet requiring 5 minutes, an individual swallowed 35 times in order to equalize pressure in the middle ear. As the descent began, a difference of pressure on the inner and outer aspects of the tympanic membrane of 30 mm. of mercury necessitated equalization of pressure. At 33, 66, and 99 feet, however, pressures of 61, 76, and 91 mm. of mercury respectively produced a comparable degree of discomfort.

Although these values are subject to considerable fluctuation, it is observed that a relationship exists between the tendency of the gas volume in the middle ear space to decrease in relation to the ambient pressure, and the necessity for equalization of pressure by voluntary opening of the auditory tube. It was necessary, for example, to equalize pressure by swallowing 17 times between 1 and 2 atmospheres pressure, and 18 times between 2 and 4 atmospheres pressure.

Theoretically, the tendency toward production of edema, hemorrhage and pain in the middle ear is the same for changes in pressure either from $\frac{1}{4}$ to $\frac{1}{2}$, $\frac{1}{2}$ to 1, 1 to 2, or 2 to 4 atmospheres. The results obtained are in accord with this consideration.

The tympanic membrane is structurally capable of moderate movement before the limit of normal excursion is reached and the cupping action on the membrane lining the middle ear and mastoid air cells begins. Rupture of a tympanic membrane when an auditory tube is blocked, requires an increase of pressure of about 5 pounds per square inch at a pressure level of 1 atmosphere.

GASEOUS DISTENTION OF THE GASTRO-INTESTINAL TRACT during ascents to high altitudes, produced by expansion of gases, may limit the altitude ascent. Gas in the alimentary tract originates largely from the nitrogen residual of swallowed air.

It is of interest to record that early in the experimental development of helium-oxygen diving the project was almost abandoned before it was recognized that in the use of a mouthpiece for helium respiration large quantities of gas were swallowed. During decompression the

rapid expansion of the trapped gas caused intense griping pain, creating the fear of viscus rupture.

Air introduced into the duodenum quickly passes through the small intestine and accumulates in the segments of the large bowel. A quantity of swallowed air will also be trapped in the stomach. At an altitude of 35,000 feet the volume will be increased four times over the volume at sea level. Uncomfortable distension is frequently experienced beginning about 20,000 feet.

Efforts toward prevention of this condition are to be directed largely to limitation of air swallowing and abstinence from food prior to flight. The use of chewing gum should be strictly interdicted prior to take-off on high-altitude missions.

SECONDARY PRESSURE PHENOMENA

FORMATION OF AIR EMBOLI IN THE BLOOD STREAM RESULTING FROM RAPID DECREASES IN PRESSURE.—Research workers interested in the effects on personnel of rapid ascents to high altitudes have considered for some time the probability of bends or gas bubble formation in the blood stream. Armstrong⁴ cites symptoms indicative of air embolism developing in individuals exposed to simulated altitudes above 30,000 feet. Symptoms described include joint pains, sudden onset of paralysis, and loss of consciousness under conditions in which adequate oxygen pressure was maintained in the lungs. Symptoms of bends in divers rapidly subjected to high altitudes have been observed in recent work at this unit. The prevention of air embolism is a major problem in military aviation.

THE NATURE OF BENDS, AIR EMBOLISM, AERO-EMBOLISM.—Atmospheric nitrogen is dissolved in the body tissues in proportion to its partial pressure in the respired atmosphere. If the atmospheric pressure is suddenly decreased, a condition is produced in which the nitrogen pressure in the body tissues is above the partial pressure in the lung air. If the degree of nitrogen supersaturation thus produced in the blood is greater than can be tolerated, bubble formation will occur and gas emboli form. While the possibility of bubble formation in tissues is imminent in animals rapidly decompressed from saturation at high pressure atmospheres, we have seen no clinical evidence to indicate that bubble formation occurs except in the blood stream.

The gas emboli are carried throughout the body by way of the blood stream. Gaseous emboli block the circulation in the same manner that air obstructs the action of a water pump. The areas of the body with the poorest collateral or total circulation are preeminently affected. Symptoms produced depend upon the location of the emboli and may appear immediately or as late as 9 to 12 hours

⁴ Armstrong, H. G.: *Principles and Practice of Aviation Medicine*. Williams and Wilkins, Baltimore, Md., 1939.

after exposure, depending upon the rate of bubble growth and the effectiveness of blood supply to the part.

The symptoms of pruritis and cutaneous rash are manifestations of bubble formation in cutaneous vessels. The substernal distress frequently felt by caisson workers and which may develop into a choking sensation and asphyxia, is indicative of emboli in the pulmonary bed. Cerebral symptoms apart from dizziness and disturbances of vision are not common. The grave danger from emboli results from their chance lodgment in the spinal cord, especially in the lumbodorsal area, with production of ischemic necrosis and the attendant symptoms of paralysis of the lower extremities, and incontinence of urine and feces.

The interesting finding of Armstrong that the spinal pressure is increased at altitudes above 18,000 feet points to bubble formation in the cerebrospinal fluid. The clinical significance of this finding has not been evaluated at this time, nor has the finding of large increases of cerebrospinal pressure been corroborated under controlled conditions in which pressure change alone is the main variable. In tests conducted in this laboratory the measured increase of cerebrospinal fluid pressure has been negligible at altitudes of 40,000 feet.

Fatigue, debilitating in severity, is one of the earliest symptoms of decompression embolism, and it may be unaccompanied by graver manifestations of bends. Usually it is of sudden onset and appears 3 to 4 hours after exposure. The resulting ennui is comparable to the malaise of influenza. Recovery usually takes place during the night's sleep. The effects are cumulative, however, and if the same exposure be undergone daily, frank bends should develop eventually. A similar condition has been observed recently following rapid ascents to high altitudes without the benefit of adequate decompression. It is believed that this fatigue is caused by the presence of bubbles insufficient in size or amount to cause pain or the graver manifestations of bends, but possibly capable of producing toxic substances by anoxic cellular destruction, or of slowing circulation and impeding venous return to the right side of the heart.

It is probable that the "X" factor described by Armstrong as an anoxemia-like effect of decreased barometric pressure, and characterized by a profound mental and physical depression, is a manifestation of fatigue due to low-grade air embolism.

ALTITUDES AT WHICH BUBBLE FORMATION MAY BE ENCOUNTERED.—Experience in diving operations indicates that the body can tolerate a pressure drop from 1 to 2 atmospheres. In terms of decreased pressure or ascent to altitudes, this drop is equivalent to a reduction in pressure from 1 to $\frac{1}{2}$ atmosphere, equivalent to an ascent from sea level to 18,500 feet. Armstrong's tests indicated that 30,000 feet is a critical level for bubble formation. It must be pointed out that

not only is the altitude reached a factor in production of air embolism but equally important is the length of time of exposure to the decreased barometric pressure at that altitude. One may make an ascent to 40,000 feet for a short period with impunity, but exposures for a period as long as 6 hours at much lower altitudes may be productive of symptoms of air embolism. The level at which bubbles form depends upon the rate of ascent analogous to the rate of decompression of divers from deep depths.

COMPOSITION OF GAS BUBBLES EVOLVED IN THE BLOOD AT HIGH ALTITUDES.—The partial pressure of gases in a bubble rapidly come into equilibrium with the gas pressures in the surrounding fluid. Table 1 indicates the partial pressures and the percentage composition of gases in a bubble present in the venous blood at sea level, and at altitudes of 27,500, 33,700, and 42,000 feet, equivalent in pressure to $\frac{1}{2}$, $\frac{1}{3}$, and $\frac{1}{4}$ of an atmosphere, respectively.

TABLE 1.—*Computed percentage composition of gas bubbles*

Altitude, feet	0	27,500	33,700	42,000
Nitrogen.....	83	48	32	-----
Carbon dioxide.....	6	18.5	24	37
Water vapor.....	6	18.5	24	37
Oxygen.....	5	15	20	-----

It is observed that the higher the altitude, the higher is the percentage composition of carbon dioxide and water vapor in the bubble, and the percentage nitrogen is consequently decreased correspondingly. This fact has particular significance in influencing the severity and amenability to treatment of altitude bends or air embolism. Should a bubble of the composition indicated in table 1 develop at an altitude of about 35,000 feet, an increase of barometric pressure brought about by a descent of a few thousand feet would probably relieve the symptoms due to the fact that a large part of the carbon-dioxide and water-vapor components of the embolus would be quickly dissipated to reduce the size of the bubble. This is in contrast to diving bends in which the embolus is composed largely of nitrogen, which requires many hours for absorption. It may be said, therefore, that the bends produced by high altitude ascents are more amenable to prompt treatment and are consequently less dangerous than are diving bends. Should the process be allowed to continue and increase in severity, however, to the point that the blood supply of vulnerable tissues is obstructed by emboli, then ischemic necrosis and irreparable damage are to be expected, identical with that produced by and described under the discussion of diving bends.

PREVENTION OF AIR EMBOLISM OR BENDS AT HIGH ALTITUDES depends upon the removal of the gaseous nitrogen dissolved in the tissues of

the body. This removal can be effected by slow ascents to high altitudes, or by removal of gaseous nitrogen from the body before the aviator begins his ascent. One method of producing such decompression is by oxygen inhalation.

We have demonstrated that the nitrogen dissolved in body tissues can be removed completely by breathing oxygen over a period of 9 hours. Figure 2 shows the results in graphic form of a typical test in which a young, well-developed man breathed oxygen for 6 hours. Curves "B" and "C" indicate the diffusion of nitrogen from the tissue solvents, water and fat. It is seen that gas diffusion from body fluids

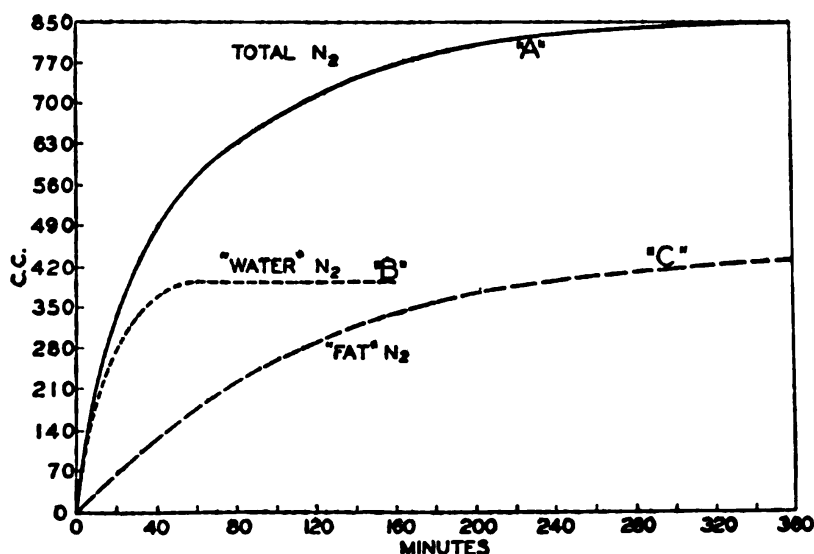


FIGURE 2.—Nitrogen elimination during breathing of oxygen.

is rapid, but for fat a period of more than 6 hours is required for complete desaturation.

The duration of oxygen breathing or the degree of body desaturation required prior to ascent varies with the altitude at which the flight is to be made and the length of time to be spent at that altitude.

Tests have been made in which the nitrogen dissolved in the body tissues has been removed and replaced by helium. Following a period of oxygen breathing for removal of sufficient helium to prevent the formation of emboli, the subjects made uneventful ascents to high altitudes.

Exercise during the first 30 minutes of oxygen inhalation prior to ascent promotes a rapid removal of nitrogen contained in the rapidly desaturating tissues. Exercise sufficient to increase oxygen consumption two and one-half times produced a doubling of nitrogen elimination during the first 10 minutes and about a 40 percent increase in elimination during the first 30 minutes.^{5 6} Following this period the

⁵ Behnke, A. R. and Willmon, T. L.: Gaseous nitrogen and helium elimination from the body during rest and exercise. *Amer. J. Physiol.* 131: 619, January 1941.

⁶ Willmon, T. L. and Behnke, A. R.: Nitrogen elimination and oxygen absorption at high barometric pressures. *Amer. J. Physiol.* 131: 633, January 1941.

measured increase of elimination was negligible. Therefore, it would appear that the value of exercise is limited to the first 30 minutes of decompression and would be of greatest value in ascents requiring little decompression. The nitrogen contained in the slowly desaturating tissues, notably the bone marrow, may not be appreciably affected by exercise.

EFFECT OF ALTERATIONS IN GAS PRESSURES

ANOXIA RESULTING FROM DECREASED OXYGEN PRESSURE IN THE LUNGS.—Anoxia means oxygen lack, and the term is applicable to any condition that retards oxidative processes in the tissues. It is to be distinguished from anoxemia or the state of oxygen deficiency in the blood. In ascents to high altitudes we are primarily concerned with anoxia consequent upon anoxemia induced by decreased oxygen pressure in the lungs.

To clarify these terms attention may be called to the anoxemia of carbon-monoxide poisoning in which, because of the high affinity of hemoglobin for carbon monoxide, the oxygen carrying power of the blood has been reduced to bring about anoxia in tissues. Anoxia not dependent upon anoxemia, but upon slowing of the circulation, may be induced by extreme chilling of the body.

A great deal of our physiological knowledge has been founded on test-chamber experiments in lieu of actual ascents in aircraft. While in the main, conditions in the laboratory have closely simulated actual flying conditions, yet it is necessary especially in controversial matters to determine whether the data were obtained in the cockpit of an airplane or within the four walls of a laboratory. In controlled laboratory tests, however, the factor of anoxemia alone can be evaluated as it affects an individual in contrast with aircraft flight which introduces such complicating variables as cold, noise, vibration, acceleration, and the strain incident to task performance.

In table 2 are shown some significant alveolar gas-pressure values incident to high-altitude ascents. The values given are pressures computed and measured in millimeters of mercury.

TABLE 2.—*Alveolar gas-pressure values incident to high-altitude ascents*

Atmosphere pressure	Altitude, feet	Alveolar gas pressure in mm. Hg.			Hemoglobin, percent O ₂ saturation
		O ₂	CO ₂	H ₂ O	
1	0	100	40	47	95
$\frac{3}{4}$	11,500	60	—	47	85
$\frac{1}{2}$	18,500	45	—	47	70
$\frac{1}{4}$	27,000	28	—	47	40
$\frac{1}{8}$	33,000	18	—	—	12

TOLERANCE TO LOW OXYGEN PRESSURES.—Healthy men at rest tolerate a reduction in oxygen pressure of about 33 percent. From table 2 it is seen that an alveolar oxygen pressure of 60 mm., reached at 11,500 feet, produces an oxyhemoglobin saturation of about 85 percent. This altitude is critical for below a level of 60 mm. the oxygen pressure in the tissues falls rapidly. In pneumonic patients recovery may not be expected if the oxygen saturation of the hemoglobin remains long below 80 percent.

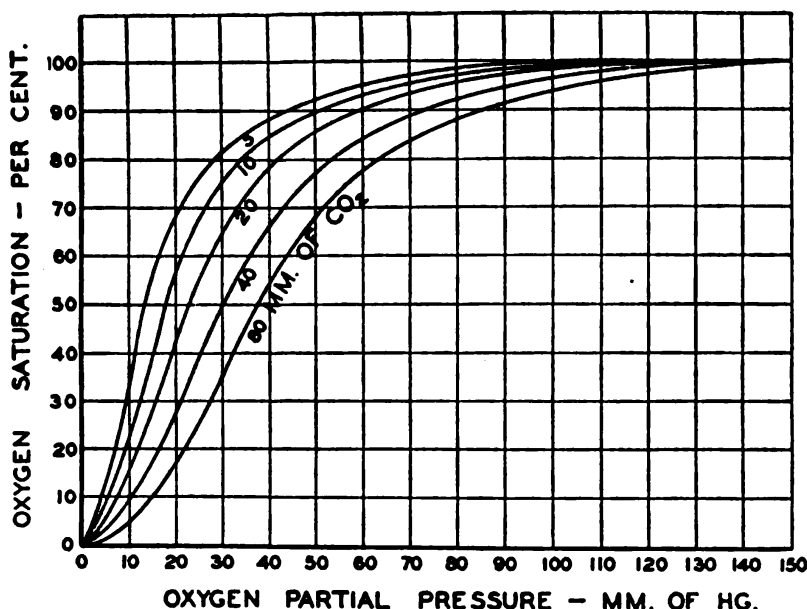


FIGURE 3.—Oxygen-hemoglobin dissociation curves. (Published in *Principles and Practice of Aviation Medicine*, Armstrong; Williams and Wilkins, Baltimore. Reproduced by permission of the publishers.)

In the oxygen dissociation curve (fig. 3), it is apparent that at a normal carbon dioxide pressure of 40 mm. and an oxygen partial pressure of 60 mm., the curve is sharply declining and the percentage drop in saturation of oxyhemoglobin outstrips the numerical value of the pressure decrease expressed in millimeters. It is not surprising that an altitude of 12,000 feet represents a rather constant "ceiling" where the majority of individuals will show marked effects of oxygen deprivation.

Individuals living at relatively high altitudes become acclimated by definite physiological adjustments.⁷ Increased lung ventilation and increased production of red blood cells and hemoglobin tend to maintain adequate oxygen uptake and transport to the tissues, while the elimination of excess bicarbonate restores the normal acid-base balance in the body. These compensatory reactions require time and are not developed by daily 7-hour exposures to altitudes of 12,000 feet.³ Instead, men subjected to these conditions develop symptoms of

⁷ Gemmill, Chalmers L.: Acclimation to high altitudes. See p. 178 of this issue.

chronic altitude sickness characterized by irritability, nervousness, insomnia and fatigue, and by occasional attacks of nausea, anorexia, and vertigo. Objectively, men may show a fall in body temperature and loss of weight. In view of these findings and similar observations made on aviators of the last war, it must be concluded that not only is tolerance to altitudes not developed, but of more importance is the fact that the critical altitude for breathing air is decreased due to deterioration of the individual.

Consideration of the effect of anoxia or tolerance to low oxygen pressure must be evaluated in terms of **degree of oxygen deficiency** and the **time factor** which includes duration and rapidity of exposure. In altitude ascents, anoxia begins as low as 9,000 feet if the exposure is prolonged; in rapid ascents anoxia terminates in coma at about 25,000 feet. At about 12,000 feet individuals may become tired, depressed or sleepy, or they may exhibit euphoria or other manifestations of emotional instability. Headache is usually a constant symptom as the altitude is increased.

Of the critical faculties, impairment of judgment is the most serious. The lack of objective ability is in marked contrast to the subjective feeling of confidence, poise, and sense of accomplishment.

These reactions, typical of anoxia induced by high altitude flight, are also observed in divers or other individuals subjected to high pressures in the range of 4 to 10 atmospheres. The cause of the abnormal reactions in divers however, is not oxygen deficiency, but what we have termed the narcotic action of atmospheric nitrogen associated with high air-pressure environments. The symptoms are immediately and almost completely abolished by substituting helium for the nitrogen in the respired gas. It is considered that the abnormal emotional and neuromuscular response resulting from exposure to anoxia or compressed air is due in each instance to depression of function of the nervous system. The marked similarity of the response induced by the anoxia and by high nitrogen pressure is striking. What is important is that able men by increased effort, "subjective reinforcement," are able to counteract the impairment and to carry out their assignment although reaction time may be greatly slowed. The less capable individual, on the other hand, loses his grasp and objective and indulges in emotional aberrations of the type commonly associated with the ingestion of alcohol.

RESPIRATION IN RELATION TO ALTITUDE.—Increased depth of breathing is a most important compensatory reaction to decreased oxygen pressure. Respiratory increase serves not only to secure better aeration of the alveoli and a more complete saturation of hemoglobin, but also to increase the effective oxygen pressure by washing out alveolar carbon dioxide. The decrease in carbon dioxide pressure in the lungs in relation to altitude is shown in table 3.

TABLE 3.—Carbon dioxide pressure in the lungs in relation to altitude

Altitude, feet	Barometric pressure mm. Hg.	Carbon dioxide pressure mm. Hg.
0.....	760	40
6,000.....	615	36
14,100.....	458	27
24,600.....	312	21

Increased respiratory depth at altitudes in good reactors produces an increase of 3 to 10 liters per minute volume of respired air. On return to ground level the breathing is usually subnormal, due to the fact that the normal carbon dioxide tension has been reduced. Normal

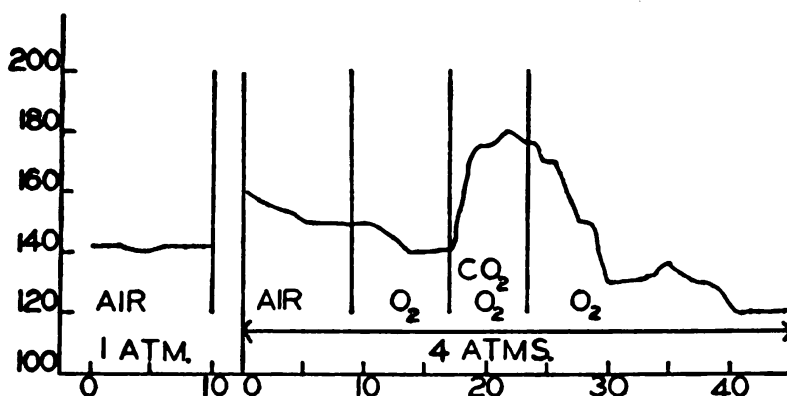


FIGURE 4.—Effect of carbon dioxide on pial arterioles. (Published in the Amer. Journ. Physiol. 111:557, 1935. Reproduced by permission of the Publishers.)

respiration awaits the accumulation in the body of carbon dioxide removed during a period of hyperventilation.

The stimulating effect on respiration of carbon dioxide concentrations up to 5 percent is well known; higher concentrations may induce narcosis. In a recent test aboard a submarine the crew breathed air containing 2 percent carbon dioxide for a period of 24 hours without discomfort except for increased respiratory minute volume in response to exercise. No harmful after effects were noted.

The important physiologic effect of carbon dioxide is dilation of cerebral vessels. These important findings as reported by Gibbs, Gibbs, and Lennox⁸ are shown graphically in figure 4, which illustrates changes in the diameter of a pial arteriole of a cat breathing a 2 percent carbon dioxide (equivalent to 8 percent carbon dioxide at 1 atmosphere) and 98 percent oxygen mixture at 4 atmospheres' pressure. Ordinate, diameter in microns; abscissa, time in minutes. This increase in the caliber of vessels and the augmentation of blood flow through the brain, appear to explain in part the stimulating effect of

⁸ Gibbs, F. A., Gibbs, E. L. and Lennox W. G.: Changes in human cerebral blood flow consequent on alterations in blood gases. Amer. J. Physiol. 111: 557, April 1935.

carbon dioxide. In higher concentrations it is conceivable that vascular dilatation may lead to congestion which might explain the headache characteristic of and following inhalation of high concentrations of carbon dioxide.

With reference to aviation, Dill⁹ observed that a concentration of carbon dioxide of 3 percent in the inspired air raised the tolerance to low pressures associated with the inhalation of air at simulated altitudes and served, for example, to ameliorate the symptoms of anoxia at 16,000 feet so that an apparent reduction in altitude to 12,000 feet was effected. Both increase of pulmonary ventilation and cerebral blood flow are to be considered as factors responsible for the beneficial effect of carbon dioxide.

In pH studies of the blood of individuals, a sharp distinction must be drawn between the effects brought about by air inhalation and those

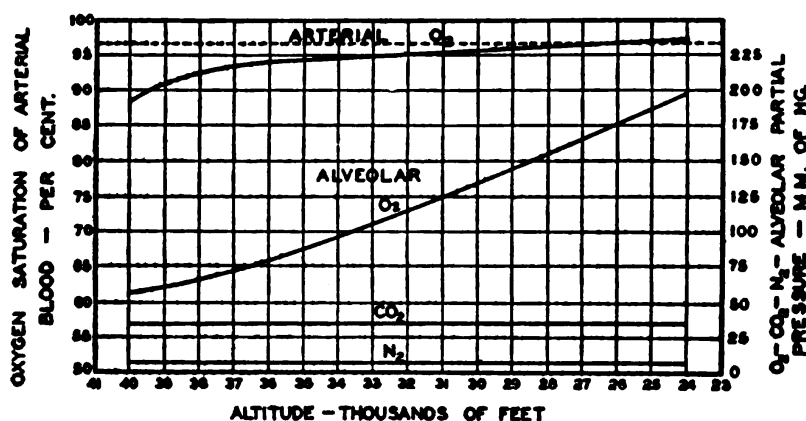


FIGURE 5.—Effects of pure oxygen on alveolar air and arterial blood at high altitudes. (Published in *Principles and Practice of Aviation Medicine*, Armstrong; Williams and Wilkins, Baltimore. Reproduced by permission of the Publishers.)

associated with the inhalation of oxygen. Armstrong and Heim² found, for example, that while breathing oxygen at altitudes between 24,000 and 40,000 feet, the alveolar carbon dioxide pressure remained constant at about 36 mm. of mercury, figure 5. The inhalation of air, on the other hand, at considerably lower altitudes would be associated with a temporary alkalosis brought about by hyperventilation, table 3.

Studies of the acid-base balance of the body at high altitudes are of great importance particularly if exposures are prolonged. Of interest is the fact that a greater loss of carbon dioxide may be expected from the body at high altitudes on the basis of greater diffusibility and elimination in the rarefied medium. At high barometric pressures on the other hand evidence points to greater difficulty in elimination of carbon dioxide.

⁹ Dill, D. B.: *Life, Heat and Altitude*. Harvard University Press, Cambridge, 1938.

ROLE OF OXYGEN IN AVIATION MEDICINE

Oxygen is the specific prophylactic and therapeutic measure for the symptoms of altitude anoxia. The early and proper administration of oxygen is the fundamental medical and physiologic procedure in connection with altitude flying. Military personnel are required at present to utilize oxygen in all flights above 15,000 feet, in flights between 12,000 and 15,000 feet of 2 or more hours duration, and in flights at or in excess of 10,000 feet for periods of 6 hours or longer. While these regulations are indicative of appreciation of the necessity for oxygen utilization at altitudes below those formerly considered necessary, it is considered that regulations should include a lower altitude-time factor range, to approach the ideal condition of maintaining a normal pressure of alveolar oxygen at all times. In connection with the need for oxygen administration, the medical officer's role is clearly outlined. The pilot, because of the insidious effects of anoxia cannot assume responsibility for determining his oxygen requirements.

The effective utilization of oxygen, however, is contingent upon adequate apparatus for administration, a problem that has not yet been solved completely.

Although Donati of Italy in 1934 reached an altitude of 47,358 feet in an unsealed cockpit plane, the maximum height that may be considered in any way practicable is limited by a safe tension of alveolar oxygen. When adequate oxygen breathing equipment is developed, this maximum height will be about 37,000 feet in unsealed cabins.

When pure oxygen is breathed, an altitude of about 33,000 feet may be reached without reducing the normal oxygen pressure in the alveoli (fig. 5). At 40,000 feet the oxygen pressure is reduced to 63 mm. or to about the same pressure that exists in the lungs at 12,000 feet when air is breathed. Breathing pure oxygen has raised the critical altitude or ceiling about 28,000 feet.

Apart from the maintenance of a normal oxygen pressure up to 33,000 feet, it should be noted that the carbon dioxide pressure in the lungs remains constant. We therefore should expect the complete elimination of all symptoms of altitude sickness up to an altitude of about 35,000 feet.

Oxygen inhalation is not without its limitations. On the basis of animal experiments it has been held that pure oxygen breathed at atmospheric tension for a few hours is toxic, producing pulmonary irritation and symptoms referable to the nervous system. There appears to be a variance between the effects of oxygen on man and animals. Experiments have been conducted by us in which 99 per cent oxygen has been breathed by man as long as 18 hours without apparent harm. Other reports have appeared in which anoxic

patients have breathed bottle oxygen more or less continually for as long as 5 days.

During the period of oxygen therapy, however, it must be noted that oxygen breathing was interrupted for varying periods from time to time, and also presumably the oxygen absorptive power of the body was impaired due to disease processes. In certain experiments we have felt that some pulmonary and nervous irritation was produced after 6 hours.

It is known that tolerance to oxygen varies among different subjects and that a form of allergy to oxygen may be produced.⁸ One individual breathed oxygen daily under increased barometric pressure for a period of several weeks without untoward symptoms. Suddenly, however, oxygen inhalation produced nausea and generalized weakness, followed by the formation of wheals, dermatographia, and flushing of the face and later dermatitis. The nausea and weakness disappeared shortly after removal of the oxygen. The dermatitis diminished over a period of 10 to 14 days but reappeared upon administration of oxygen. Remission of symptoms was hastened by administration of histaminase.

Our data with regard to oxygen toxicity at high altitudes are scant. Since from tests at high pressures it appears that oxygen pressure rather than percentage is the factor governing toxicity, then pure oxygen at a pressure of 456 mm. (60 percent of 760 mm.), can be breathed indefinitely without injury.

In any proposed oxygen tests environmental temperature and the relative presence of food in the alimentary tract are important factors relative to appearance of symptoms of oxygen toxicity as pointed out by Campbell.¹⁰

We have considered only some of the physiologic effects associated with high altitude flying. Yet it should be clear that the unaided flyer of today is helpless compared with his predecessor in the last war, due to demands made by increased speed, acceleration, altitude, and maneuverability of modern aircraft.

In addition to the dangers of anoxemia and aero-embolism, there is the problem of combating temperatures as low as $-60^{\circ}\text{F}.$, a problem that may yet prove to be the most formidable. There are also the problems of affording protection against noise, vibration, and the fatigue and exhaustion incident to the strain of complicated task performance.

Engineering accomplishments have done a great deal to promote flying safety but the urgent problem now is to maintain on the basis of applied physiology a constant *interne milieu* for the aviator in the face of tremendously adverse environments.

⁸ Campbell, J. A.: Oxygen poisoning and tumor growth. Brit. J. Exp. Path. 18: 191, June 1937.

Upon the ability of medical officers and civilian scientists to accomplish this task will depend the life of the aviator in terms of flying hours, barring a purely military casualty.

SUMMARY AND CONCLUSIONS

1. Primary pressure phenomena in the range of $\frac{1}{4}$ to 15 atmospheres are manifested by a cupping action on the membranes lining the middle ear and sinus spaces when obstruction in channels opening into these spaces prevents equalization of pressure.

2. An unreported clinical entity is described in which delayed pain, congestion, and hemorrhage affect the middle ear following oxygen inhalation during high altitude pressure changes. These symptoms develop during sleep when oxygen absorption in the middle ear spaces tends to create a differential not equalized by voluntary opening of the auditory tubes.

3. Aero-embolism, essentially identical with deep-sea diving bends, develops following ascents to high altitudes if adequate decompression is not previously administered.

4. Additional studies are required regarding the full role of carbon dioxide in high altitude ascents and in regard to acid-base balance in body tissues.

5. A prime physiological principle to be emphasized in aviation is that alveolar oxygen pressure should approach normal at all times to prevent anoxia, acute and chronic, which may first make its insidious appearance at altitudes as low as 9,000 feet.

ACCLIMATION TO HIGH ALTITUDES¹

A REVIEW OF PHYSIOLOGICAL OBSERVATIONS

By Lieutenant Commander Chalmers L. Gemmill, Medical Corps, United States Naval Reserve

Information concerning the chronic effect of high altitude on man has come from several sources. First, organized parties of physiologists have spent several weeks living at high altitudes and making observations on themselves during that time. Notable examples of this type of research are the parties organized by Haldane,² Barcroft,³ and Dill⁴ and Keys.⁵ Their reports contain the results of first-class

¹ From the Department of Physiology, Johns Hopkins University, Baltimore, Md.

² Douglas, C. G., Haldane, J. S., Henderson, Y., and Schneider, E. C.: *Physiological observations made on Pikes Peak, Colo., with special reference to adaptation to low barometric pressures.* Phil. Trans. Roy. Soc. London B, 203: 185, 1913.

³ Barcroft, J., Binger, C. A., Bock, A. V., Duggart, J. H., Forbes, H. S., Harrop, G., Meakins, J. C., and Redfield, A. C.: *Observations upon the effect of high altitude on physiological processes of the human body carried out in the Peruvian Andes, chiefly at Cerro de Pasco.* Phil. Trans. Roy. Soc. London, B., 211: 351, 1923.

⁴ Dill, D. B.: *Life, Heat, and Altitude. Physiological Effects of Hot Climates and Great Heights.* University Press, Cambridge, 1933.

⁵ Keys, A.: *Physiology of life at high altitudes; international high altitude expedition to Chile, 1935.* Scient. Monthly, 43: 289, October 1936.

physiological research.* Second, information has come from laboratories placed at high altitudes, such as those on Monte Rosa (14,960 feet) and on the Jungfrauoch (11,320 feet). Very careful and systematic work has been done in these laboratories on the effect of high altitude on man. Third, chambers have been constructed in which man has lived for various periods of time at a reduced oxygen tension. Barcroft,⁶ for example, remained in such a chamber for 6 days, and more recently Matthews⁷ was subjected to low oxygen tension for 5 days in a similar room. Experiments of this type are not as spectacular as those involving living at a high altitude, but they may be more carefully controlled, especially as to external temperature, food, and fatigue. Fourth, information has been obtained from parties organized primarily for climbing and not for physiological research. While these observations are not the result of carefully controlled conditions as those of the other groups, they are of interest, because they have been obtained at higher altitudes. Expeditions have climbed to a height of 28,000 feet on Mount Everest and have stood on top of Nanda Devi, a peak 25,645 feet in altitude. Additional information is given in table 1 concerning the altitudes of the Himalayan peaks and the heights that have been climbed. It was on such climbing trips that numerous physiological observations have been made.

TABLE 1.—*Heights that men have climbed*⁸

Name of mountain	Location	Altitude in feet	Height climbed
Mount Everest.....	Nepal Himalaya.....	29, 002	28, 000
K ₂	Karakoram.....	28, 250	26, 000
Kangchenjunga I.....	Nepal Himalaya.....	28, 146	25, 256
Nanga Parbat.....	Punjab Himalaya.....	26, 620	25, 256
Gasherbrum I.....	Karakoram.....	26, 470	22, 960
Nanda Devi.....	Garhwal Himalaya.....	25, 645	25, 645
Kamet.....	do.....	25, 447	25, 447

Although the older literature concerning high altitude studies made by physiologists has been reviewed by Loewy⁹ and the newer results by Keys,¹⁰ much of the physiological information obtained by the climbing parties has not been incorporated into these summaries. It is, therefore, the purpose of this review to collect some of these physiological observations of climbing parties acclimatized to high altitudes. The data reported by organized parties of physiologists will be used to

* Barcroft, J., Cooke, A., Hartridge, H., Parsons, T. R., and Parsons, W.: Flow of oxygen through the pulmonary epithelium. *J. Physiol.* 53: 450, May 1920.

⁶ Barcroft, J., Elliott, R. H. E., Fraser, F. R., Herkel, W., Matthews, B. H. C., and Talaat, M.: Case of deficient acclimatization to low oxygen pressure. *J. Physiol.*, 82: 369, October 1934.

⁷ Bates, R. H.: Five Miles High. Dodd, Mead and Co., New York, 1939.

⁸ Loewy, A.: Physiologie des Höhenklimas. Julius Springer, Berlin, 1932.

¹⁰ Keys, A.: Die Wirkung des Höhenklimas und die akklimatisierungsprozesse in Grosser Höhe. *Ergebnisse der Inn. Med. und Kinderheilkunde*, 54: 585, 1938.

augment the results obtained by the climbing parties only when the latter's observations are insufficient.

RESPIRATORY CHANGES

The most obvious change in man acclimatized to high altitude is an increase in respiratory activity. A good description of this is given by Somervell,¹¹ a member of the first Everest expedition. He relates that at 26,000 feet his respiratory rate was 55 and that 5 respirations were needed for every step upwards and that at 28,000

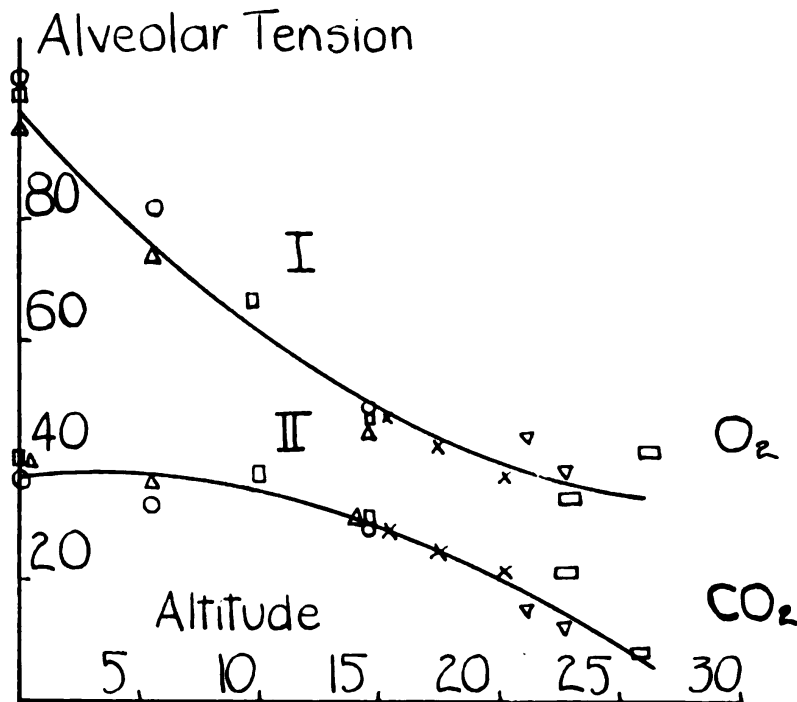


Figure 6.—Relationship between alveolar carbon dioxide and oxygen tension and altitude. Ordinates, alveolar tension in mm. Hg.; abscissa, altitude in thousands of feet. Curve I, alveolar O₂ tension; curve II, alveolar CO₂ tension. Data from Dill x (12); Ward o (13); Barcroft □ (2); Haldane Δ (1); Warren ▽ (11) and Greene □ (10).

feet 7 to 10 respirations were required per step. This increase in ventilation causes the composition of alveolar air to approximate that of atmospheric air. There is, therefore, a decrease in the tension of carbon dioxide and an increase in that of oxygen in the alveolar air spaces of the lungs. Since alveolar air is in equilibrium with arterial blood, corresponding changes occur in this blood. Somervell,¹¹ Greene,¹² and Warren¹³ have collected and analyzed alveolar air in

¹¹ Somervell, T. H.: Note on the composition of alveolar air at extreme heights. *J. Physiol.*, 66: 283, September 1925.

¹² Greene, R.: Observations on the composition of alveolar air on Everest. *J. Physiol.*, 82: 481, November 1934.

¹³ Warren, C. B. M.: Alveolar air on Mount Everest. *J. Physiol.* 96: 34P, 1939.

their climbs on Mount Everest. Unfortunately, the first observer collected his samples in rubber bags. Since he did not make his analyses until several days later, the slow diffusion of the gases into and out of the rubber bag makes his determinations untrustworthy. Greene ¹² and Warren ¹⁸ used evacuated tubes and carefully controlled their collections of alveolar air.

A summary of their results with a comparison of other observations made at lower altitudes is given in figure 6. The highest elevation at which a sample was obtained was 25,700 feet. At this altitude

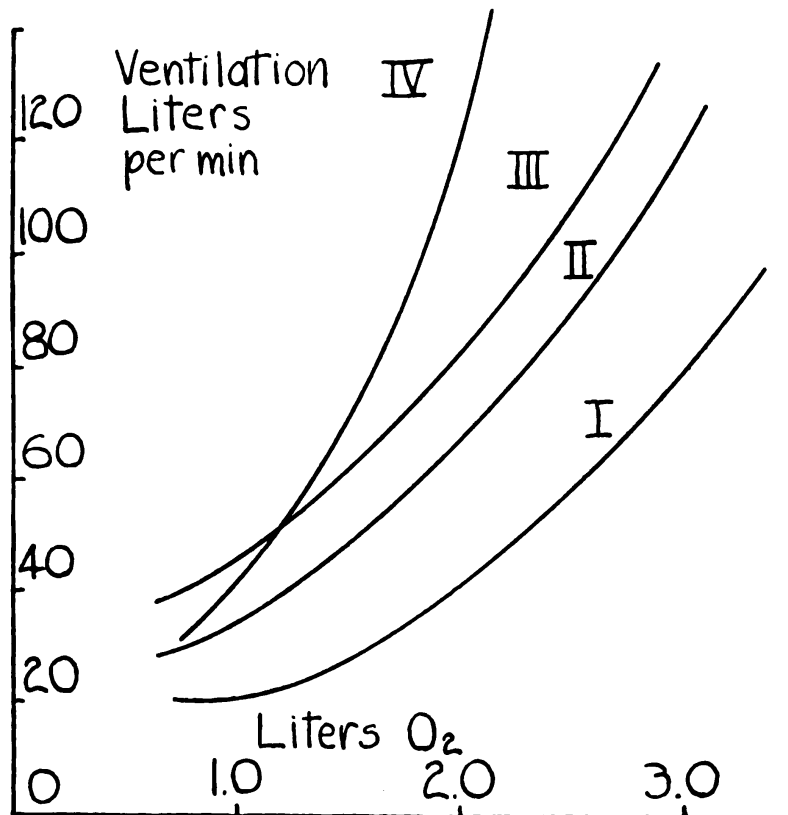


FIGURE 7.—Relationship between oxygen consumption during work in liters of oxygen per minute and total ventilation in liters per minute at various altitudes. I, sea level; II, 9,219 feet; III, 12,000 feet; IV, 17,500 feet. Data from Christensen (15).

the alveolar oxygen tension was 43.0 mm. Hg, and the carbon dioxide tension was 9.2 mm. Hg. It may be seen in the figure that the carbon dioxide tension falls to a very low level in order that an oxygen tension is maintained compatible with life. It is obvious that the respiratory control based on a normal carbon dioxide tension of the arterial blood has ceased to function at high altitudes. A drop in the carbon dioxide tension to 9.2 mm. Hg would cause apnea at

sea level, whereas at a high altitude this tension of carbon dioxide is associated with marked hyperventilation.

Muscular work at high elevations is associated with a much greater respiratory response than at sea level. Hingston ¹⁴ states that above 19,000 feet the slightest exertion, such as tying a shoe lace, is associated with marked respiratory distress. Christensen ¹⁵ has measured accurately the ventilation associated with work at various altitudes. His results are given in figure 7. During the production of a certain amount of work demanding 2 liters of oxygen per minute, there was a respiratory ventilation of 40 liters per minute at sea level, whereas at 17,520 feet a similar amount of work required a ventilation of 120 liters per minute.

TABLE 2.—*Vital capacity at high altitudes* ¹⁶

Height (subject)	8,800	16,600	17,700	21,000	16,000	13,800
C. W.-----	4.3	4.3	4.0	4.1	4.1	4.3
E. K.-----	4.0	3.8	4.0	3.9	4.0	4.2
E. W.-----	4.3	4.6	4.8	5.0	5.0	5.2

With this hyperventilation at high altitudes there is very little change in the vital capacity. Some of the results recorded by Warren ¹⁶ are given in table 2.

The ability to hold the breath diminished with altitude. Richter, ¹⁷ the physician with the Dyhrenfurth expedition, reports that at 7,020 feet a member of his party could hold his breath 73 seconds, but at 20,079 feet this value had decreased to 15 seconds. Hingston ¹⁷ obtained similar results. Their findings are given in figure 8. In a similar way the ability to blow a column of mercury to a height of 40 mm. and to hold it at this height is diminished. Hingston ¹⁸ observed that he could hold the mercury elevated for 45 seconds at sea level, but at 21,000 feet the best he could do was 15 seconds.

CIRCULATORY CHANGES

Many mountain climbers have recorded their pulse rates at high altitudes. The basal pulse rate does not change until heights above 15,000 feet are reached and is only slightly elevated above this height. Hingston ¹⁸ records that Norton's resting pulse rate at sea level was

¹⁴ Hingston, R. W. G.: *Physiological difficulties in the fight for Everest, 1924*. E. Arnold and Co., London, 1925.

¹⁵ Christensen, E. H.: *Sauerstoffaufnahme und respiratorische Funktionen in grossen Höhen*. Skand. Archiv. f. Physiol., 76:88, 1937.

¹⁶ Warren, C. B. M.: Cited by Rutledge, H. in *Everest, the Unfinished Adventure*. Hodder and Stoughton, London, 1937.

¹⁷ Richter, H.: Cited by G. O. Dyhrenfurth in *Arztliche Beobachtungen, Himalaya*. Scherl, Berlin, 1931.

¹⁸ Hingston, R. W. G.: Cited by Barcroft, J.: *The Respiratory Function of the Blood*. University Press, Cambridge, 1925.

40, but at 27,600 feet it was only 60. There is, however, a much greater change in pulse rate at high altitudes when exercise is attempted than at sea level. For example, Warren¹⁶ showed that the increment in the pulse rate was greater with greater altitudes when the subject rose from a lying to a standing position, figure 9. Hartmann's^{19 20} results demonstrated a similar effect. Using a dynamometer to standardize the exercise, he found that the work curve

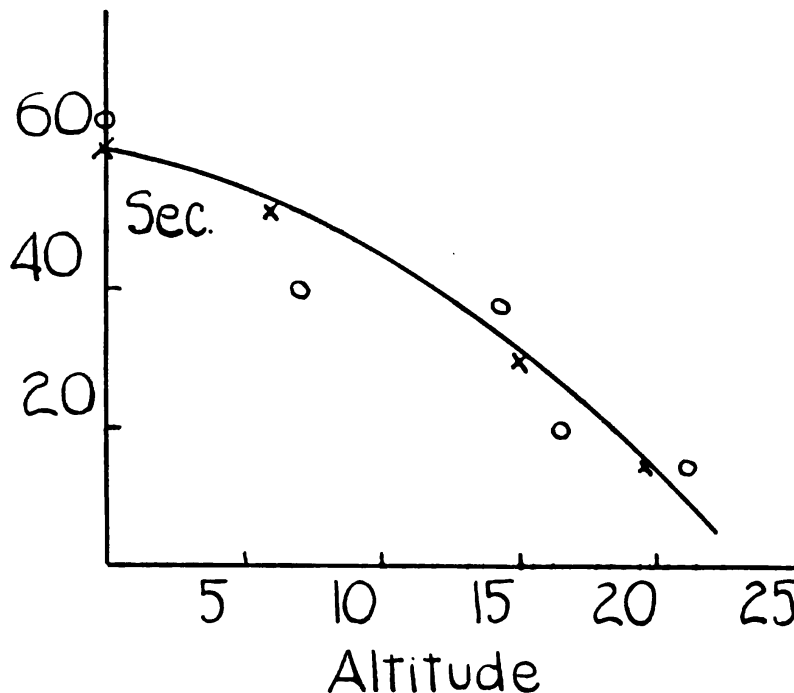


FIGURE 8.—Time for holding the breath at various altitudes. Ordinate, minutes; abscissa, altitude in thousands of feet. Data from Richter x (17) and Hingston o (14).

was a straight line and that the basal curve did not rise until heights over 15,000 feet were reached, figure 10. This observer was killed in a snow avalanche in the Himalayas. Somervell¹¹ states that his pulse rate during climbing at 26,000 feet was 180 beats per minute. Kraus²¹ records that his basal pulse rate at sea level was 60; at 22,966 feet, 88. During climbing it rose to 144. Another member of the party, Bauer, had a basal pulse rate of 76 at sea level, 92 at 22,966 feet and 112 during climbing at that altitude.

¹⁹ Hartmann, H.: Experimentell physiologische Untersuchungen auf der Deutschen Himalaya-Expedition, 1931. *Zeit. für Biol.* 23: 391, 1932-33.

²⁰ Hartmann, H.: Die Wirkung grosser Höhen auf den Organismus vor und nach erfolgter Anpassung. *Verhand. deut. Gesell. für Inn. Med.* 47 Kongress, 48, 1935.

²¹ v. Kraus, K.: Cited by P. Bauer. In *Kampf um den Himalaja*. Knorr and Hirth, München, 1931.

Hingston¹⁸ measured the blood pressures of the members of the 1924 Everest Expedition at various heights from sea level to 21,000

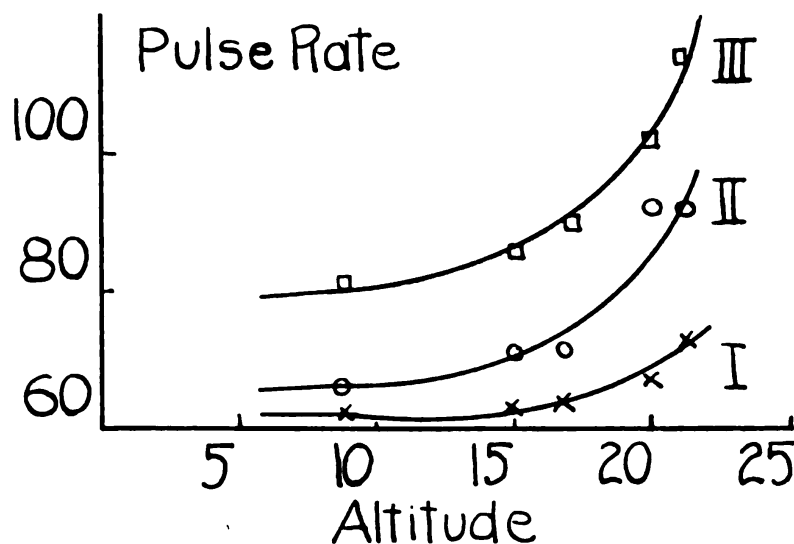


FIGURE 9.—Relationship between pulse rate and altitude. Basal pulse rate I; sitting pulse rate, II; and standing pulse rate, III. Ordinate, pulse per minute; abscissa, altitude in thousands of feet. Data from Warren (16).

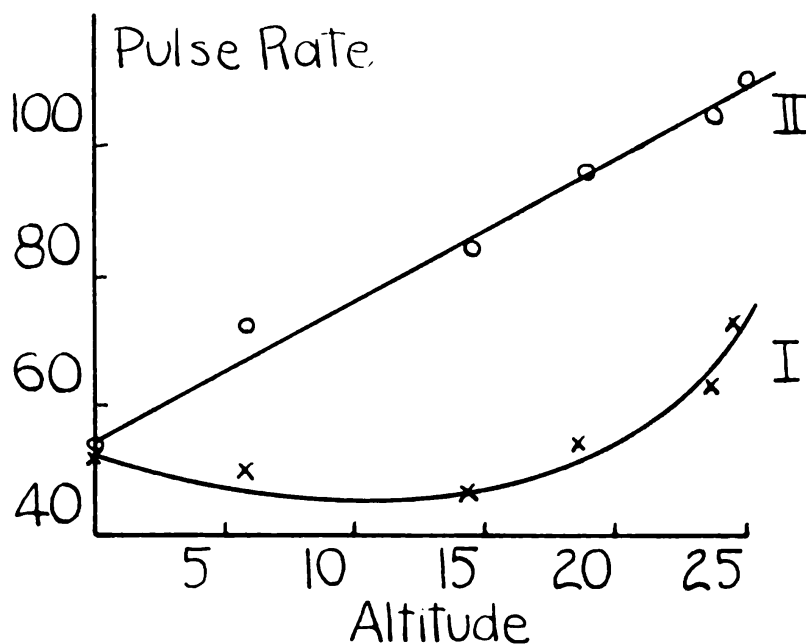


FIGURE 10.—Relationship between basal and working pulse rate and altitude. I, Basal pulse rate; II, pulse rate after standardized exercise. Ordinate, pulse per min.; abscissa, altitude in thousands of feet. Data from Hartmann (18).

feet and reports that there was no significant change in blood pressure associated with altitude.

Numerous studies have been made on the number of red blood cells and the hemoglobin content of the blood at various altitudes. The most complete series of determinations on red cell concentration were made by Hingston²² who spent several months in the Western Himalayas and the Pamir plateau and reached an altitude of 18,203 feet. Hingston's results are given in figure 11. His series is note-

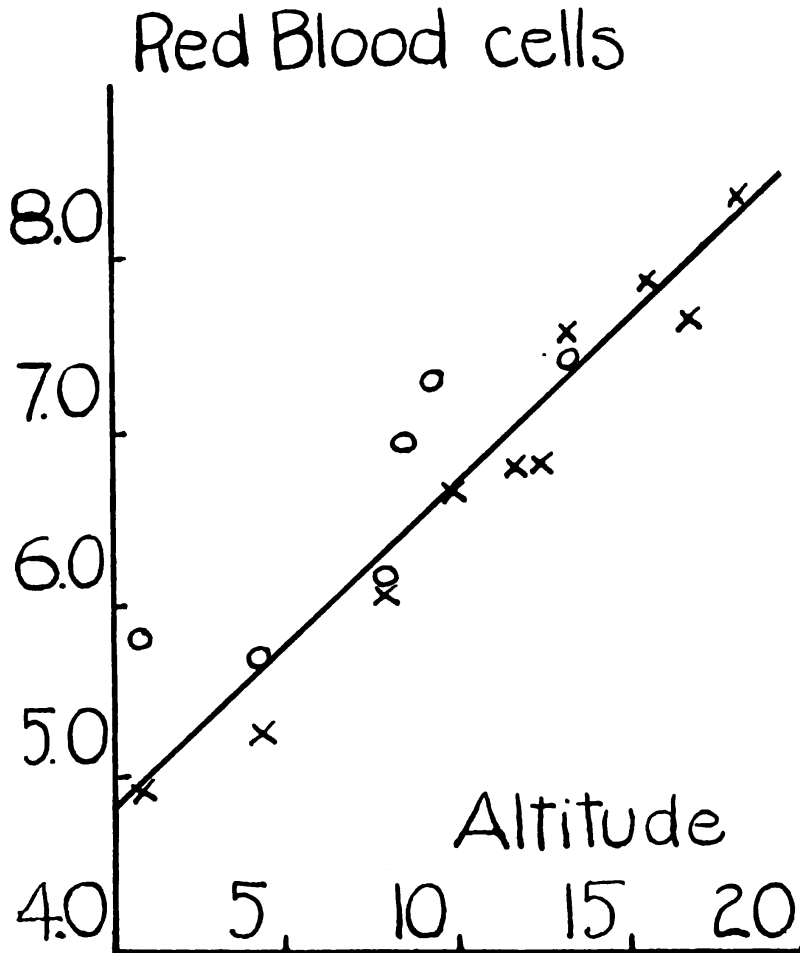


FIGURE 11.—Relationship between red blood cells in millions per cubic millimeter and altitude in thousands of feet. Data from Hingston (20). Determinations during ascent x; during descent o.

worthy, since he studied the return of the red cell concentration to normal during his descent. The majority of physiological observations made by other observers involve only the transition from sea level to high altitude. The physiological changes produced by going from high altitude to sea level have been neglected by physiologists.

Two series of determinations on hemoglobin content of the blood at various altitudes are given in figure 12. Warren's¹⁶ results ob-

²² Hingston, R. W. G.: Physiological changes at high altitudes and their relation to mountain-sickness. *Indian J. M. Research*, 9: 173, July 1921.

tained on Mount Everest show very little change in hemoglobin percentage until 15,000 feet was reached. At this point there was a sharp break in the curve and the concentration increased rapidly. The results of FitzGerald²³ show a steady rise with each increment in altitude. Her results were obtained on groups of miners living at various altitudes in Colorado. Warren's results represent an individual's response while FitzGerald's give an average of many determinations at various altitudes.

It is of interest to calculate the oxygen content of the blood when an individual is at 20,000 feet and has 130 percent hemoglobin con-

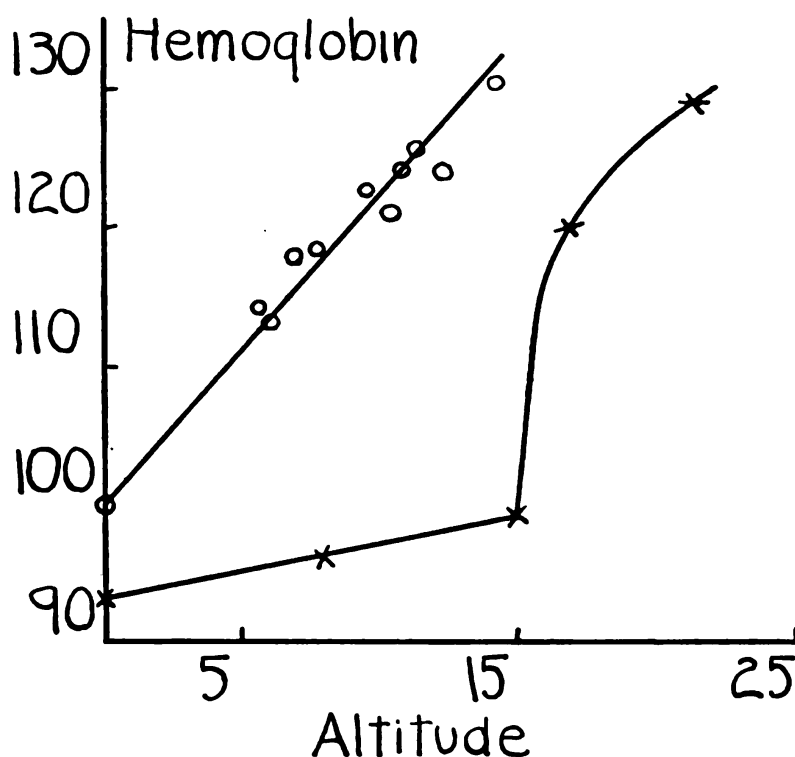


FIGURE 12.—Increase of hemoglobin with altitude. Ordinate, hemoglobin per cent; abscissa, altitude in thousands of feet. Data from FitzGerald o (21) and Warren x (16).

centration. At sea level, saturated blood carries 21 ml. of oxygen per 100 ml. of blood. At a concentration of 130 percent of hemoglobin, saturated blood would carry 27.3 ml. of oxygen. However, at 20,000 feet the oxygen tension of arterial blood is approximately 40 mm. Hg, and the blood at this tension is only 80 percent saturated with oxygen. Therefore, the blood would carry 80×27.3 or 21.84 ml. of oxygen per 100 ml. of blood. This amount compares favorably with the amount of oxygen carried at sea level, but compares unfavorably with the tension which is much less. The reduction in tension

²³ FitzGerald, M. P.: The changes in the breathing and the blood at various high altitudes. *Phil. Trans.* 203: 351, 1913.

has reduced the driving power used in pushing the oxygen from the capillaries into the tissues.

CONCLUSIONS

Man has lived for 10 days at 23,000 feet and has climbed to 28,000 feet. The few changes which have been studied in man acclimatized to such altitudes have been in the fields of respiration and circulation. These findings have demonstrated remarkable adaptations. Many additional observations are needed before a complete story may be made as to man's adaptation to such heights. For example, nothing is known of the reaction of the cells of the body following acclimatization. Does cellular respiration change in order that the cells might oxidize their metabolites more efficiently under reduced oxygen tension? This is just one of the many problems of acclimatization that have not been answered at the present time.

SUMMARY

A review is given of some physiological studies on man following acclimatization to high altitudes. These results were obtained for the most part by parties organized to climb mountains. These findings are of interest, for they have been obtained at higher altitudes than those observed by groups of physiologists organized primarily to study the effect of high altitude on man.

DUTIES OF THE DENTAL OFFICER IN NAVAL COMBAT

By Commander Francis G. Ulen, Dental Corps, U. S. Navy

Concurrent with and as a result of the present rapid expansion of the naval establishment, there has been some confusion in the minds of recently commissioned dental officers and some reserve dental officers who have been ordered to active duty as to the nature and extent of their responsibilities. Many of these officers have not and in all probability will not have the advantage of attending the course that is given for the indoctrination and training of younger officers at the Naval Dental School. Some will serve at larger dental activities ashore under the direction of dental officers of the regular Navy who have had long experience in the naval service and are thoroughly cognizant of the duties of the dental officer and of all laws and regulations that concern his activities. Here by precept and example they may acquire a knowledge of what is required of them. Others, not so fortunate, may have no such preparation prior to active duty at sea. It is for the information of the latter that this paper is written.

Generally, all available information relative to the routine duties of the naval dental officer may be acquired by a diligent perusal of Navy Regulations, The Manual of the Medical Department, General Orders,

Uniform Regulations, Circular Letters of the Bureau of Medicine and Surgery, *et cetera*. However, very little specific information is contained in these publications relative to the duties of the dental officer in combat.

The Manual of the Medical Department states "The Dental officer shall be assigned to a battle-dressing station"¹ and that "Dental officers * * * shall familiarize themselves with the duties to be performed at such stations."² The Landing Force Manual specifies that "Medical and dental officers of the landing force shall be given training in the pistol courses."³ Thus it follows that the duties which a naval dental officer may be required to perform, particularly in combat, extend beyond the field of the routine practice of dentistry.

A letter, whose authorship is generally attributed to John Paul Jones, in commenting upon the qualifications of the navy line officer of that time states, "It is by no means enough that an officer of the navy should be a capable mariner. He must be that, of course, but also a great deal more." To paraphrase this, it might be said that it is by no means enough that a naval dental officer be a capable dental surgeon. He must be that, of course, but also a great deal more. He must be cognizant of the basic principles of modern naval action as they affect the function of the battle-dressing station if he is to efficiently carry on as an officer in charge of or acting as an assistant at such a station. These principles are so well known as to be axiomatic to the older and more experienced medical and dental officers. The material for this paper has been drawn almost exclusively from articles which have been written by these officers. However, repetition here is considered to be justified in order that this information may be brought to the attention of those dental officers who have had limited duty or no duty in combatant ships.

To understand the extent and restriction of activities of the medical department during a naval engagement, the dental officer must know that it is essential that the watertight integrity of the ship and the fire power as delivered by the ship's batteries must be maintained regardless of the sick and wounded.⁴ He must know that the maximum contribution of the medical department toward maintaining the ship at full battle efficiency consists of the treatment of the wounded as soon as practicable in order that they may be returned to their battle stations. He must realize that the prompt relief of pain not only serves to combat shock but also has a salutary effect upon the maintenance of morale, for whereas in action ashore the killed and wounded may be left behind the scene of combat by the

¹ Manual of the Medical Department, U. S. Navy, 1938. Par. 884.

² Manual of the Medical Department, U. S. Navy, 1938. Par. 231.

³ Landing Force Manual, U. S. Navy, 1938. L. F. M. 19-9 (2).

⁴ U. S. Fleet Letter No. 23L-33 (revised).

shifting of the battle line, at sea they remain underfoot, where the sight of their wounds and the sound of their groans cannot fail to adversely affect the morale of their shipmates. Thus he will realize that the effectiveness of the men at their battle stations may be maintained, in part at least, by the removal of the dead and wounded at the earliest practicable time, and that such removal must be accomplished with the minimum interference with the activities of the station at which they are found.

He must know that medical department materiel and personnel are so dispersed as to render the maximum aid for the wounded as is consistent with the maintenance of the ship at battle efficiency and why certain medical department personnel may be exposed under hazardous circumstances.

An evaluation of the problem of prompt treatment of battle casualties will lead him to the conclusion that first-aid instruction to non-medical personnel as well as to hospital corpsmen is essentially a continuous and important process. It is continuous in that the rapid turn-over of personnel in the Navy as a whole and in the ship to which he is attached results in the loss of men so trained. It is important in that the Hospital Corps alone is numerically too small to supply first-aid men to all battle stations. The importance of this training for nonmedical personnel has been brought to the attention of all medical and dental officers by recent circular letters of the Surgeon General.

As an officer of the Navy, the dental surgeon must realize that during action, it will be his task to deport himself in such a manner as to instill confidence in the men under his direction and to maintain order and discipline under conditions that will try the courage and patience of the bravest. When below and removed from the scene of battle he and his subordinates will await, with such calmness and fortitude as they may be able to muster, a lull in the battle at which time it may be possible for them to actively engage in the relief of the wounded.

As an officer of the medical department, he should study statistics of personnel casualties of former naval actions to familiarize himself with maximum casualty rates in individual ships, casualty rates percent of complement, the ratio of killed to wounded and particularly the percentage of wounds which may be expected to occur about the face and head. As a result of his study, he will more fully comprehend just what may be required of him professionally during and after a modern naval engagement.

He must be familiar with the location, equipment, and function of the battle-dressing stations, collecting stations, repair parties, and first-aid boxes. He must understand the battle organization and

system of internal communication of the ship as they affect the function of the battle-dressing stations. He must know what parts of the ship are to be served by each of the battle-dressing stations, traffic routes from each of these stations to those parts of the ship which they serve, and alternate routes if such exist. The possession of this information will enable him to arrive at a more accurate estimate of emergencies which may arise during action and of corrective measures which must be taken in case of these emergencies.

A short time ago the then Surgeon General of the Navy wrote:

In order to promote efficiency at the battle-dressing stations, this Bureau is considering the advisability of adding a course to the curriculum of the Naval Dental School, designed to adapt the professional training of dental officers to the problem of prompt treatment of battle casualties. Should this prove feasible, proficiency in such duties would be made a subject of examination for promotion in the Dental Corps.

Such instruction is now being given at the Naval Dental School. The following is a short résumé of the subject with which the dental officer should familiarize himself in his professional capacity as an officer of a battle-dressing station:

1. Sorting of wounded relative to the urgency of treatment required.
2. Chemical agents: tactical classification, physiological classification, properties of each, symptoms produced, general and specific treatment of gas casualties, methods of degassing and decontamination after action, and protective clothing.
3. Hemorrhage: types to be expected and first-aid treatment to control.
4. Wounds: care of open and closed wounds and method of disposal.
5. Fractures and dislocations: method of splinting and reducing.
6. Fractures of the skull and injuries of the spine: probable prognosis and methods of handling.
7. Shock: symptoms and treatment of.
8. Methods of transportation of wounded.
9. Artificial respiration: resuscitation of the electrically shocked and the apparently drowned.
10. Types and methods of battle bandaging.
11. Conservation of medical department supplies and their distribution during battle.

TREATMENT OF CARDIOVASCULAR EMERGENCIES

By Lieutenant Commander Arthur M. Master, Medical Corps, United States Naval Reserve

INTRODUCTION

Acute situations requiring immediate treatment arise more frequently in diseases of the heart and arteries than in disease of any other system. Furthermore, heart diseases now lead all others in incidence, chiefly as a result of the increased span of life. The physician should, therefore, be thoroughly acquainted with the most recent advances in cardiac therapy. It is our belief that many lives can be saved and others made more comfortable if certain simple, therapeutic principles are observed.

HEART FAILURE

Heart failure may be divided into two types, left ventricular and right ventricular. The former is much more common, since the most frequent types of heart disease produce strain and enlargement of the left ventricle, such as essential hypertension, coronary artery disease, aortic stenosis and insufficiency due to rheumatic, luetic or congenital heart disease, acute and chronic nephritis, coarctation of the aorta, etc. Rarely acute left heart failure is seen in mitral stenosis. When the left ventricle becomes insufficient, there is back pressure in the left auricle and pulmonary veins. This produces an increase in intrapulmonary pressure, resulting in pulmonary congestion, dyspnea on exertion and orthopnea. Later paroxysmal nocturnal dyspnea and cardiac asthma may set in, culminating in attacks of frank pulmonary edema.

In the milder forms of acute left heart failure there are numerous rales in the chest. These may be of all types, from the fine crackles resembling those found in early pneumonia to the coarse rales of pulmonary edema, and frequently are indistinguishable from those of bronchitis or bronchial asthma. The second pulmonic sound is accentuated. The arm-to-lung circulation time is prolonged, and the vital capacity diminished. In the more acute forms, ashy cyanosis and Cheyne-Stokes respiration may be present, and the chest may be full of bubbling rales typical of pulmonary edema. Frequently there is evidence of shock with peripheral failure, that is, the skin is cold and clammy, the pulse rapid and thready, and the blood pressure unobtainable.

As the degree of left ventricular failure increases, secondary right ventricular heart failure may appear. Primary right heart failure is less common and is associated chiefly with mitral stenosis and pulmonary disease. With the onset of right heart failure there is back pressure in the superior and inferior vena cavae; the cervical veins become engorged and pleural effusion, enlargement of the liver, ascites and edema of the extremities may appear, producing the picture of dropsy. Cyanosis increases but dyspnea may diminish. The arm-to-lung time and venous pressure are increased.

When pulmonary edema is present, morphine, 0.015 gram (gr. $\frac{1}{4}$), should be administered subcutaneously. If this does not suffice it may be repeated in 15 minutes. Occasionally it has to be administered intravenously in the dosage of 0.005 gram (gr. $\frac{1}{2}$). Simultaneously tourniquets are placed on the extremities (venostasis), reducing the venous return to the heart. Atropin is not of use in the writer's opinion. If relief is not obtained, aminophyllin, 0.12 gram (gr. 2), is given by vein, very slowly, and may be repeated. If the pulmonary edema persists, venesection should be performed unless the veins are collapsed, as in coronary occlusion; 400 to 1,000 cc. of

blood may be removed within 5 to 10 minutes. It has been shown that this increases the vital capacity several hundred cc. Occasionally strophanthin, 0.3 milligram (gr. $\frac{1}{200}$), by vein is effective, but it should not be used in coronary occlusion or if digitalis has been given previously. We do not administer hypertonic dextrose intravenously, having found it without beneficial effect, and it may do harm by increasing the work of the heart. Unless the condition of the patient improves very rapidly, oxygen should be administered. This may be done by means of a tent, nasal catheter, or the recently introduced BLB mask. By means of the latter it is possible to administer up to 100 percent oxygen, whereas with the tent the usual percentage is 50 or 60. However, a cool temperature can be maintained in the tent, a desirable feature in warm weather. A relatively low oxygen percentage is obtained with the nasal catheter, but if neither of the other two methods is available it is of value. Recently the administration by BLB mask of 100 percent oxygen under increased pressure has been advocated for pulmonary edema.

When the acute episode has been successfully combated, the routine measures in the treatment of subacute and chronic heart failure are instituted. These are employed regardless of whether the failure is of the left or right ventricle. They include complete rest in bed, limitation of fluid and salt, a low calorie diet, beginning with the Karrel diet in severe cases, and sufficient sedation to produce restful sleep. Adequate catharsis is maintained. Mercurial diuretics are invaluable. 1 to 2 cc. of mercupurin may be given by vein or muscle every 4 to 5 days, preceded by and concomitantly with adequate doses of acidifying drugs, such as ammonium chloride, 4 to 6 grams (gr. 60-90) daily. The mercurials may also be given in suppository form. Full digitalization is also instituted. The dosage required depends upon the weight and sex of the patient, although there are individual variations in response to the drug. In general one cat unit, 0.1 gram (gr. $1\frac{1}{2}$), is required per 10 pounds body weight in males. Therefore, the usual digitalization dose for a man of 150 pounds is 15 cat units, 1.5 grams (gr. $22\frac{1}{2}$). If digitalis has not been administered previously it may be given in divided doses such as eight cat units, 0.8 gram (gr. 12), the first day, four cat units, 0.4 gram (gr. 6), the second, and two cat units, 0.2 gram (gr. 3), thereafter, until the ventricular rate has fallen to 60-70 beats per minute or nausea or vomiting threatens. A daily maintenance dose is then given, usually 1 cat unit, 0.1 gram (gr. $1\frac{1}{2}$). Occasionally, venesection is helpful if the venous pressure is high. Conflicting opinions have been expressed concerning the indications for mercurial diuretics and digitalis in either right or left ventricular failure. They are effective in both. It is very frequently not recognized that the effect of mercurial diuretics is markedly reduced or even prevented by the following drugs: Mor-

phine or its derivatives, codeine, quinidine, amidopyrine, adrenalin and ephedrin. Therefore these drugs should be avoided or used as little as possible when diuretics are being used. The xanthine group of drugs such as theobromine, 0.3 gram (gr. 5), theophylline, 0.3 gram (gr. 5), etc., four times a day by mouth has not proven as useful as the mercurial diuretics in heart failure, although they are more effective when given by rectal instillation or suppository. For example, aminophyllin suppository, 0.5 gram (gr. 7½) 3 times a day. Frequently a very effective diuretic is urea 30 grams (gr. 450) three times a day. It should not be given when azotemia is present. Unfortunately many patients find this drug disagreeable to take. We have found beer and grapefruit juice relatively good vehicles but it is necessary to avoid imbibing large amounts of fluid with the drug.

In neglected cases the degree of right heart failure may be advanced until there are collections of fluid in the chest or abdomen or, rarely, in the pericardium, which require aspiration. Occasionally such a collection causes acute distress; thus we have recently seen two cases of urgent dyspnea resulting from the sudden formation of a pleural effusion. Rarely the dropsical state results in massive edema of the extremities which is not relieved by the ordinary methods; in such a case the edema fluid can sometimes be removed by inserting Southey tubes into the legs.

Occasionally one encounters what seems to be a severe stage of heart failure of undetermined etiology which does not respond to the ordinary therapeutic measures. In such instances one should think of myxedema and vitamin B deficiency, the beri-beri heart. In the latter condition the heart is very flabby and dilated and, unlike any other type of heart failure, the circulation time is not prolonged. Prompt and complete relief can be obtained in mild or moderately severe cases with vitamin B, particularly thiamine chloride 50 milligrams intravenously daily. Later 5-milligram doses may be administered orally. The myxedema heart usually improves with thyroid extract; if a large pericardial effusion is present, paracentesis may be required.

Heart failure is not common in the acute infections except in the face of antecedent heart disease. However, we have observed it in typhoid fever, diphtheria with myocarditis, etc., in persons with hearts normal prior to the illness. The therapy of heart failure in infections is the same as that given above except that digitalis should be withheld unless other measures are unsuccessful. Furthermore, digitalis should not be given routinely in infections, for example pneumonia, in the effort to prevent heart failure.

Heart failure, particularly of the left ventricle, is frequently attended by Cheyne-Stokes respiration. It occurs chiefly on the verge of falling asleep and following the administration of morphine. It responds

quickly to 0.12 gram (gr. 2) aminophyllin by vein and coramine, 1 to 2 cc. of the 10-percent solution. Caffein sodio-benzoate, 0.5 gram (gr. 7½), may be effective for Cheyne-Stokes respiration caused by morphine. Inhalation of oxygen with 5 to 7 percent carbon dioxide also is very helpful.

ARRHYTHMIAS

Changes in rhythm often produce alarming symptoms including palpitation, precordial pain, vertigo, vomiting, syncope, collapse. Occasionally, these are severe enough to simulate cardiac catastrophes such as coronary occlusion and acute heart failure, and, indeed, may initiate these. On the other hand they may occur in the absence of heart disease. In such cases the cause may be an infection, a toxin such as tobacco or alcohol, or Graves' disease. This should always be searched for and eradicated.

SINUS TACHYCARDIA.—This is usually found in association with such conditions as heart failure, Graves' disease, infections like tuberculosis or pneumonia, intoxications, etc. Occurring alone it is rarely important clinically. It may set in paroxysmally and produce palpitation. In addition, several young adult women have been observed whose pulse is persistently 120 to 140 without obvious cause. There is no therapy except sedation, but an attempt should be made to determine and eradicate the cause, e. g., heart failure or infection.

SINUS BRADYCARDIA.—This is not of clinical importance except in the rare instances in which the rate drops to 40 or less. When this occurs the patient may complain of faintness. An example of this is the person with a sensitive carotid sinus in whom fainting may be associated with sudden slowing of the heart. In simple bradycardia atropin, 0.6 milligram (gr. 1/100), and ephedrin, 0.05 gram (gr. 3/4) by mouth or adrenalin, 1 cc. of the 1:1000 solution subcutaneously, may be used when symptoms are present.

PREMATURE BEATS.—These are auricular, nodal or ventricular, depending upon the focus of origin. Premature beats are exceedingly common, occurring in normal as well as diseased hearts. In persons sensitive to tobacco they may be associated with smoking. Another common cause is distension of the stomach. Usually they occur singly and thus produce no symptoms. Not infrequently, however, they are multiple and very numerous, resulting in palpitation which at times is violent. If sedation is ineffective in such cases, quinidine sulphate may abolish the arrhythmia. A test dose of 0.2 gram (gr. 3) is given and if the patient is not sensitive, this dose may be given every 2 to 3 hours for 1 to 2 days. If a satisfactory result is obtained, the drug may be maintained several times daily to prevent the recurrence of the premature beats. Occasionally atropin, 0.6 milligram (gr. 1/100) three times a day by mouth, is efficacious when quinidine has failed.

AURICULAR FIBRILLATION.—This may be paroxysmal or permanent. Both types usually occur in damaged hearts but may appear in normal persons. It is most common in arteriosclerotic and rheumatic heart disease with heart failure. A common cause is Graves' disease, and it occurs in infections such as rheumatic fever, pneumonia, etc.

The paroxysmal form usually sets in abruptly and is accompanied by palpitation and sometimes precordial pain or pressure. The apical rate is usually between 120 to 180 and there is often a pulse deficit. If the heart is normal, heart failure does not appear until the arrhythmia has persisted for days. When cardiac disease is present, the rapid rate may induce heart failure or shock after a short period.

In treating a paroxysm of auricular fibrillation, as well as episodes of auricular flutter and tachycardia, it is well to remember that these arrhythmias are apt to remit spontaneously. Therefore, it is best not to administer specific drugs for several hours unless heart failure or shock is present. During this period sedatives or narcotics may be given. If the fibrillation is still present after a period of observation, and heart failure has not appeared, quinidine should be given. After a test dose of 0.2 gram (gr. 3) to make sure the patient is not sensitive to the drug, this amount is given every 2 hours. If there is no change in rhythm, the dose should be doubled and, if necessary, given every hour. The drug must be taken day and night, since it is rapidly excreted. Occasionally, it is necessary to raise the dose to 0.6 gram (gr. 9) every 2 hours, a total of 6 gram (gr. 90) a day. If the arrhythmia is still unchanged after 24 hours, the drug should be discontinued. When sinus rhythm has been restored, the amount of quinidine is gradually reduced and a maintenance dose of 0.2 gram (gr. 3) three times a day is continued to prevent recurrent episodes. If heart failure is present in the acute attack digitalis should be administered instead of quinidine in order to slow the ventricular rate. Digitalis does not always induce sinus rhythm. It may be necessary to digitalize the patient rapidly, giving 8 cat units, 0.8 gram (gr. 12), the first day and 6 cat units, 0.6 gram (gr. 9), the second day. If the patient has not received digitalis prior to the attack and the degree of heart failure is severe, it is frequently advisable to give strophanthin, 0.3 milligram (gr. 1/200), or digitalis 4 cat units, 0.4 gram (gr. 6), intravenously. This may be followed by smaller doses of strophanthin, 0.1 milligram (gr. 1/600), or digitalis 2 cat units, 0.2 gram (gr. 3). When the ventricular rate has been slowed, the digitalis is taken by mouth, 1 to 2 cat units, 0.1 to 0.2 gram (gr. 1½ to 3).

In chronic auricular fibrillation the ventricular rate should always be controlled by digitalis. In Graves' disease the auricular fibrillation, whether paroxysmal or persistent, rarely is affected by digitalis or quinidine. However, if the basal metabolism is lowered with Lugol's solution, 2/3 cc. (min. 10) three times a day, the arrhythmia frequently

disappears or becomes amenable to quinidine or digitalis therapy. Patients with auricular fibrillation or flutter complicating Graves' disease should be subjected to thyroidectomy after proper preparation with Lugol's solution. In the acute infections auricular fibrillation is usually transitory.

AURICULAR FLUTTER.—Like auricular fibrillation this arrhythmia may occur in normal or abnormal hearts, usually the latter. It is usually paroxysmal but occasionally becomes permanent. The auricular rate is 260 to 360 beats per minute. The ventricular rhythm in auricular flutter may be regular, with a usual rate of 150 to 180. It can be slowed by carotid sinus pressure but this does not abolish the arrhythmia. However, the ventricular rate frequently is irregular and resembles auricular fibrillation clinically. As a rule the two can be distinguished on physical examination by the observation of rapid venous pulsations in the neck corresponding to the rate of the flutter waves.

If a paroxysm of auricular flutter does not remit spontaneously, digitalis or quinidine may be used. We have favored the former drug, particularly when there is heart failure, and have found it effective in more than half the cases. Usually rapid and complete digitalization is required to restore sinus rhythm. We administer 6 to 8 cat units, 0.6 to 0.8 gram (gr. 9 to 12), at once and repeat in divided doses in 12 to 24 hours. If the patient is vomiting because of heart failure and is unable to tolerate the digitalis by mouth, 4 cat units, 0.4 gram (gr. 6), may be given intramuscularly or 2 cat units, 0.2 gram (gr. 3), intravenously. In the transformation of auricular flutter to sinus rhythm with digitalis there is often an intermediary stage of auricular fibrillation which occasionally persists. If this happens the digitalis should be discontinued and sinus rhythm may set in spontaneously. If digitalis is not successful, quinidine may prove effective; 0.2 gram (gr. 3) may be given every 1 to 2 hours for 6 doses and this course may be repeated after several hours with a dose of 0.4 gram (gr. 6). Sometimes auricular flutter persists in spite of all these measures and the patient must be maintained on digitalis as in chronic auricular fibrillation in order to keep the ventricular rate between 60 and 70 beats per minute. In addition, digitalis may transform the flutter to fibrillation in which case the ventricular rate is more easily controlled.

When the episodes of auricular flutter recur frequently an attempt should be made to prevent them by the use of quinidine or digitalis. The dose of the quinidine is 0.2 to 0.3 gram (gr. 3 to 4½) four times a day. Rarely it may be necessary to increase the dose or the frequency of administration in order to give as much as 2 gram (gr. 30) a day. If this is not effective, digitalization should be carried out and a maintenance dose continued. (See auricular fibrillation.)

PAROXYSMAL TACHYCARDIA.—As in the case of premature beats paroxysmal tachycardia may be auricular, nodal, or ventricular in origin.

Auricular and Nodal Tachycardia.—These are considered together since they cannot be distinguished clinically and are treated alike.

It is characteristic of a bout of tachycardia that it begins and ceases abruptly. The ventricular rate is absolutely regular and is usually somewhat more rapid than in auricular flutter, as a rule 180 to 220 beats per minute. Frequently it can be differentiated from auricular flutter by the absence of flutter waves in the cervical veins. Furthermore, it is very responsive to vagal influence. Pressure on the eyeball or carotid sinus, if successful, abolishes the tachycardia completely, whereas in flutter it merely slows the ventricular rate temporarily. Auricular tachycardia is frequently functional or neurogenic in origin, occurring in normal hearts as well as in diseased ones. It may be associated with such exciting factors as smoking, drinking, infection, gastric distension, Graves' disease, and substances producing allergic reactions.

Paroxysmal auricular or nodal tachycardia is particularly apt to be evanescent. Furthermore, various maneuvers stimulating vagal tone may abolish an episode. This has been learned through experience by most of those subject to recurrent attacks. They have found that squeezing or straining the neck, holding the breath, coughing, vomiting, eating, lowering the head over the bed, etc., may terminate the paroxysm and if relief does not occur, the physician should exert eyeball or carotid sinus pressure. The right carotid sinus is more sensitive than the left. Vomiting may be induced with syrup of ipecac, 4 to 8 cc. (drams 1 to 2) by mouth, combined with vagal pressure. Occasionally these maneuvers are unsuccessful and the physician must resort to drugs. Mecholyl (acetyl-beta-methyl choline), quinidine or digitalis may be used. When properly administered mecholyl has usually proven effective. It is injected subcutaneously, the initial dose being 25 to 30 milligrams (gr. 5/12 to 1/2) which may be repeated in 20 to 30 minutes. Its effect may be enhanced by massaging the site of injection and pressing the carotid sinus. Some persons react to mecholyl with severe nausea, vomiting, diarrhea, salivation, etc., and, therefore, when giving the drug, atropin, 1 milligram (gr. 1/60), should be at hand since, given intravenously, it immediately relieves these symptoms. Also the patient should be flat in bed to prevent syncope and a tourniquet may be applied above the site of injection to prevent further absorption. One must remember that the effect of mecholyl is inhibited by quinidine, which should be used only after mecholyl has proven unsuccessful. The manner of administration of quinidine in auricular tachycardia is the same as in auricular flutter; 2 to 3 grams (gr. 30 to 45) may be required to restore sinus

rhythm. If shock or heart failure is present 0.3 to 0.5 gram (gr. 5 to 7½) quinine dihydrochloride may be given intravenously; it is obtainable in ampoule form, 0.3 gram (gr. 5) dissolved in 10 cc. saline. The injection must be given slowly; otherwise it may result in untoward reactions. It is rarely necessary to use the intravenous route to administer quinidine. If quinidine has failed digitalis should be tried, full digitalization being carried out rapidly by mouth or vein as in auricular flutter. If there are frequent recurrences of the tachycardia, their frequency may be diminished with quinidine or digitalis, as in auricular flutter. Also the factors precipitating the attack should be avoided.

Ventricular tachycardia.—This is not a common arrhythmia. Although occasionally observed in the absence of heart disease, it is almost always associated with severe myocardial damage, particularly recent or old coronary occlusion. It is sometimes possible to differentiate it clinically from auricular tachycardia by a slight irregularity in rhythm and a variation in the intensity of the apical sounds. In one exceptional case this was sufficiently marked to simulate auricular fibrillation.

Efforts at vagal stimulation are ineffective in ventricular tachycardia but it responds readily to quinidine. However, it too, may remit spontaneously and the drug should not be resorted to immediately unless the condition of the patient demands it. Usually giving quinidine by mouth, 0.2 gram (gr. 3) every hour, as already described, is sufficient, but occasionally the condition of the patient may require giving the drug by vein. 0.3 to 0.5 gram (gr. 5 to 7½) quinine dihydrochloride is injected slowly and repeated if necessary. The arrhythmia may cease promptly.

HEART BLOCK.—This arrhythmia may be divided into *partial* and *complete* block. Partial block includes the two stages, simple P-R prolongation and dropped beats, and does not give rise to clinical symptoms except in the rare instances where sufficient beats are dropped to lower the ventricular rate below 40 beats per minute. In that case the therapy is the same as that employed in complete heart block. In complete block a dissociation exists between the auricles and ventricles, the latter beating at their own idioventricular rate, which is usually between 16 and 40 beats per minute. The slow rate, particularly if it appears suddenly, or asystole, which may occur, produces cerebral anemia which is manifested by Stokes-Adams seizures, including vertigo, syncope, convulsions or coma. This may occur also when partial block abruptly changes to complete block. The Stokes-Adams syndrome is a very serious complication, and usually portends a fatal issue within several years.

The treatment of complete heart block is unsatisfactory. The only drug which can be depended upon is adrenalin. During the Stokes-

Adams seizure it may be life-saving, one-half to one cc. of the 1:1,000 solution being given subcutaneously, or a lesser amount directly into the heart in extreme cases with prolonged asystole. If the seizures recur, the adrenalin should be repeated subcutaneously regularly every few hours. Occasionally it has been found necessary to maintain a slow, continuous intravenous injection of 1:100,000 strength at 2 cc. (30 drops) per minute. Another drug which may increase the ventricular rate and prevent Stokes-Adams seizures is aminophyllin administered by vein in a dose of 0.12 gram (gr. 2) several times a day. 100 percent oxygen may be effective. When the acute condition has subsided ephedrin sulphate, 0.05 gram (gr. $\frac{1}{4}$), or paradrin, 0.01 gram (gr. $\frac{1}{4}$), may be given orally periodically. Barium chloride is ineffective.

VENTRICULAR FIBRILLATION.—The Stokes-Adams syndrome is not always associated with complete heart block; rarely the cause is ventricular fibrillation. This can be determined only by means of the electrocardiogram. When such a mechanism exists quinine dihydrochloride, 0.3 gram (gr. 5), should be given by vein and adrenalin and ephedrin must be avoided. 20 cc. of a 10 percent solution of magnesium sulphate has been given successfully. Ordinarily ventricular fibrillation is preterminal, but it may be caused by excessive digitalis and by trauma or electric shock.

PRECORDIAL PAIN

One of the commonest symptoms confronting the practitioner is acute pain in the region of the heart. Before attempting to treat this it is essential to determine whether the pain is cardiac in origin and, if it is, the specific type of heart lesion. There are innumerable conditions which may simulate acute cardiac pain; the chief of these are pneumonia, pleurisy, pneumothorax, pulmonary embolus, spondylitis, neuritis, herpes zoster, diaphragmatic hernia, ruptured peptic ulcer, acute cholecystitis, renal calculus, etc.

NEUROCIRCULATORY ASTHENIA

There is a large group of cases with precordial pain in which no organic basis can be found. Among these are the frankly neurotic persons and the group designated neurocirculatory asthenia (soldier's heart, effort syndrome). The latter may present not only severe pain but tachycardia, diminished vital capacity and exercise tolerance and changes in the electrocardiogram simulating mitral stenosis (that is, right axis deviation, large P waves, and T_{2-3} inversion). The treatment is reassurance and sedation, and, for severe cases, rest. In military life the "hardening" of this type of soldier or sailor should be gradual. He should be given special attention. His physical exertion should be minimal at first, and slowly increased.

If it is concluded that the pain originates in the heart, in persons under 30 the cause is probably rheumatic heart disease. Over the age of 30 one must also distinguish between angina pectoris due to coronary sclerosis or leutic aortitis, coronary insufficiency, and coronary occlusion. Although angina pectoris and coronary occlusion are associated with coronary sclerosis, they differ otherwise both clinically and pathologically. In angina pectoris there is a temporary ischemia which does not result in anatomic alterations in the heart muscle; there are no constant changes in the heart sounds, blood pressure, temperature, and electrocardiogram, and heart failure is absent. The pain is usually relieved by nitroglycerin, 0.3 to 0.4 milligram (gr. $\frac{1}{160}$ to $\frac{1}{200}$). Coronary occlusion, however, results in a greater or lesser area of myocardial infarction. Heart failure, shock, gallop rhythm, pericarditis, a drop in blood pressure, fever, etc., usually appear. The electrocardiogram presents a typical progressive pattern, characterized by RS-T elevations and q waves. In coronary insufficiency the duration and degree of interference with the coronary flow is greater than in angina pectoris. As a result, scattered areas of necrosis occur in the heart muscle. A clinical syndrome similar to coronary occlusion may be present. However, they can be differentiated in two ways. Whereas the onset of coronary occlusion is independent of any external factor, coronary insufficiency is usually associated with some exciting cause, such as acute hemorrhage, shock, heart failure, tachycardia, aortic stenosis, probably effort, etc. Also, unlike coronary occlusion, the electrocardiogram presents depression of the RS-T segment and q waves do not appear. Each of these three conditions is treated differently from the others.

ANGINA PECTORIS.—An attack of angina pectoris as a rule responds promptly to nitroglycerin. This is best administered under the tongue, the dose being 0.3 to 0.4 milligram (gr. $\frac{1}{160}$ to $\frac{1}{200}$). As a rule this small dose is as effective as a larger one which may produce headache and vertigo; in any case, it can be repeated if necessary. A number of persons are sensitive to nitroglycerin and the reaction may be less severe with the smaller dose. If the pain is not relieved by nitroglycerin one should suspect that coronary occlusion and not angina pectoris is present. Nitroglycerin may be taken many times a day over a long period of years without any decrease in its effectiveness. It may also be of prophylactic value, diminishing the number of anginal attacks when taken regularly and preventing an attack if taken prior to effort. If nitroglycerin is not at hand or has not relieved the pain, whiskey may be effective or even morphine required. When the anginal syndrome is persistent, that is, status anginosus is present, strict bed rest should be prescribed, since it may be the precursor of acute occlusion. If the latter is ruled out, the performance of a paravertebral sympathetic nerve block should be considered. **A**

word may be said about the severe shoulder pain which may accompany angina pectoris or follow coronary occlusion. For this large doses of vitamin B₁, thiamin chloride 5 milligrams three times a day, are being given. The treatment of angina pectoris associated with aortic insufficiency in rheumatic heart disease is similar to that due to coronary artery disease. It is of interest, however, that in these cases the pain is relieved by walking.

CORONARY OCCLUSION.—Although occasionally coronary occlusion is attended by little or no pain, the latter is characteristically excruciating at the onset and is frequently associated with a greater or less degree of peripheral collapse. Because of the pain and the presence of upper abdominal tenderness and spasticity, the differential diagnosis from acute abdomen, particularly ruptured peptic ulcer and acute cholecystitis, not infrequently presents itself. Obviously it is extremely important to make the correct diagnosis, since the treatment of the acute abdomen is usually surgical. The presence of gallop rhythm, drop in blood pressure, pericarditis, pulmonary congestion, or electrocardiographic changes indicates coronary occlusion.

If the diagnosis of coronary occlusion is suspected, the patient is immediately put to bed. Nitroglycerin, digitalis and strophanthin are avoided and in fact are harmful. If the pain is severe, morphine, 0.015 gram (gr. $\frac{1}{4}$) should be given and repeated in 15 minutes if necessary. Morphine is somewhat more effective when given intravenously, the initial dose being 0.005 to 0.006 gram (gr. $\frac{1}{2}$ to $\frac{1}{10}$). An excess of morphine should be avoided because of its constipating effect and the possibility of vomiting. Very rarely even large doses of morphine do not control the pain and aminophyllin, 0.12 to 0.24 gram (gr. 2 to 4), should be given intravenously very slowly. Occasionally 100 percent oxygen is effective. In extreme cases it may be necessary to inject sodium amytal, 0.2 to 0.5 gram (gr. $3\frac{1}{2}$ to $7\frac{1}{2}$), intravenously or to give paraldehyde, 4–8 cc. (drams 1 to 2), rectally. When the acute pain has subsided, small doses of codeine, 0.015 to 0.03 gram (gr. $\frac{1}{4}$ to $\frac{1}{2}$) by mouth, usually suffice to prevent recurrences.

The treatment of shock in coronary occlusion differs from that used in other types of shock because one wishes to avoid the administration of large amounts of fluid intravenously. Frequently the shock which accompanies the acute pain at the onset of the attack disappears when relief of the pain has been attained with morphine. If this does not occur, aminophyllin, 0.12 gram (gr. 2) intravenously, may help, this dose being repeated if necessary, and oxygen should be given. In extreme cases it may be necessary to give frequent small doses (5 minims) of adrenalin. One such patient who was moribund but recovered also received a slow, continuous intravenous drip of 5 percent dextrose.

The general treatment of coronary occlusion consists of complete rest in bed, excellent nursing care, the avoidance of visitors and a low calorie diet. During the first few days the patient may not take more than a few hundred calories daily. If there is nausea or vomiting, sips of carbonated water and bits of cracked ice may be helpful. Toward the end of the first week the intake is usually up to 800 calories and this is maintained for several weeks, depending upon the condition of the patient. This regime diminishes the work of the heart and gives the infarct a chance to heal. It also reduces gastrocardiac reflexes and as a result the tendency to precordial pain is lessened.

Heart failure, chiefly of the left ventricle, is very common in coronary occlusion which may set in with an attack of pulmonary edema. Usually, however, the heart failure is not severe during the first few days and gradually disappears. Sometimes it increases in intensity and requires treatment. A cardinal principle in the therapy of heart failure in coronary occlusion, as in any acute disease such as rheumatic fever or pneumonia, is that digitalis should not be used, unless the usual measures fail, such as limitation of fluid and salt, acidifying drugs and mercurial diuretics. The danger of digitalis lies not only in its toxic effect on the heart muscle directly but also in the tendency to produce ventricular tachycardia in the presence of infarction. When the drug is used, digitalization should not be rapid, the full dose being reached in 4 or 5 days. The treatment of pulmonary edema, cardiac asthma and Cheyne-Stokes respiration in coronary occlusion is otherwise the same as already described.

All the arrhythmias are very common in coronary occlusion and are apt to be transient, particularly auricular tachycardia. Even complete heart block may remit spontaneously. Therefore specific drug therapy should be delayed 8 to 12 hours unless there is evidence of incipient heart failure. The treatment of the arrhythmias is the same as when coronary occlusion is not present except that quinidine should be given preference over digitalis in auricular flutter and adrenalin is to be used with caution in complete heart block. Much has been written concerning the frequency of ventricular tachycardia in coronary occlusion and it has been suggested that quinidine be administered routinely early in the attack to prevent this arrhythmia. However, the incidence of this tachycardia has been only 0.5 percent in almost 1,000 personally observed cases of coronary occlusion in which digitalis was not administered, and some of the attacks ceased spontaneously.

In addition to heart failure and arrhythmias, coronary occlusion may be complicated by rupture of the heart and embolism. The former accident is much rarer than is ordinarily believed. Although usually fatal within a short time, when death is delayed it may be indicated by evidence of pericardial effusion. There is no treatment,

but the incidence of rupture may be diminished by not allowing the patient out of bed prematurely.

Mural thrombosis in the heart very commonly accompanies cardiac infarction, accounting for the high incidence of embolism in the lung, brain, kidney and peripheral arteries. The treatment of embolism will be discussed elsewhere, but we may emphasize here the importance of conservative therapy in coronary occlusion. Operation should be performed in the course of acute coronary occlusion only if absolutely necessary, since the prognosis is poor and the danger of subsequent embolization considerable.

CORONARY INSUFFICIENCY.—The clinical picture has been described previously. The management of the acute emergency is similar to that of coronary occlusion. In addition one should treat the exciting factor such as heart failure, shock tachycardia anemia, etc. For example, if there has been acute hemorrhage one or more small transfusions of 200 to 300 cc. may be advisable. If heart failure or shock is present, it should be relieved.

RHEUMATIC HEART DISEASE

During the active stage of rheumatic fever three common emergencies may arise: Acute pericarditis, congestive failure, and various arrhythmias. The latter two conditions should be treated as already outlined in the acute stage of coronary occlusion, withholding the use of digitalis if possible. In this connection, one should suspect rheumatic activity whenever the ventricular rate in auricular fibrillation cannot be controlled with digitalis.

ACUTE PERICARDITIS may be associated with very severe precordial or epigastric pain or distress, relieved only by morphine. Ordinarily, however, large doses of amidopyrin, 0.6 gram (gr. 10) every 2 to 4 hours, are effective. This drug should not be given with barbiturates and an occasional blood count should be taken to detect any fall in leucocytes. It is best to delay tapping the pericardium unless the effusion is large and increasing rapidly and is associated with progressive dyspnea and heart failure, due to cardiac tamponade. A considerable amount of fluid may be present in the pericardium without symptoms and is frequently resorbed spontaneously. In withdrawing fluid from the pericardium several routes may be used. Using a trochar, we consider it best to insert the needle in the fifth left intercostal space anteriorly above the upper border of the rib, just within the border of dullness. The needle should be advanced slowly in order to prevent touching the heart. If the cardiac pulsations are felt, the needle should be withdrawn a short distance. One should not make unnecessary attempts to obtain fluid since the heart may be punctured. When fluid is obtained, it should be withdrawn slowly. Alternative points for tapping the pericardium are just below the

border of the xiphoid process and below the angle of the left scapula. In tuberculous pericarditis several authors have advised the reinjection of air after the removal of the fluid, the volume of air being about half that of the fluid removed. Mention must be made of the presence of pericardial effusion in myxedema; if this is not relieved by thyroid extract, aspiration should be performed.

MITRAL STENOSIS.—Persons with mitral stenosis frequently present urgent problems. When it is long-standing, auricular fibrillation or flutter is apt to set in suddenly, particularly if congestive failure has been present. Also pulmonary edema occurs occasionally. The treatment of these have already been outlined in the consideration of heart failure and the arrhythmias.

BALL VALVE THROMBUS.—Auricular thrombi are frequently associated with heart failure and auricular fibrillation and, when the stenosis is extreme, a thrombus may obstruct the valve almost completely so that little blood is passed into the ventricle. A characteristic picture of acrocyanosis follows: the tip of the nose, the lobes of the ears and the fingers and toes become cold and markedly cyanotic. There is usually severe dyspnea and frequently shock. The treatment is supportive; oxygen is given and the usual measures taken to relieve the congestive failure. Morphine, 0.015 gram (gr. $\frac{1}{2}$), is helpful if the patient is restless.

EMBOLISM.—It is common for an auricular thrombus to enter the greater or lesser circulation, resulting in pulmonary or peripheral emboli. The latter may lodge in the brain, kidney, spleen, the bifurcation of the aorta or its branches. When embolism occurs in an artery that is surgically within reach, the advisability of removing the embolus is to be considered. It has been our experience that in many cases conservative medical therapy is successful. This includes prompt administration of morphine, 0.03 gram (gr. $\frac{1}{2}$), or papaverin, 0.04 gram (gr. $\frac{1}{4}$), to prevent spasm of the collateral circulation. They may be injected intravenously in doses of 0.005 to 0.006 gram (gr. $\frac{1}{10}$ to $\frac{1}{2}$). Alcohol by mouth may also be of use. If there is a tendency to gangrene the pavaex machine should be tried. In one case the severe pain attending peripheral embolism was relieved by maintaining the leg at a temperature of 36° F. This also delayed the progress of gangrene. Two other measures advocated are paravertebral sympathetic nerve block and the injection of aminophyllin directly into the vessel proximal to the site of the embolus. However, if the patient is seen within several hours of the onset and the site of the embolism is accurately determined, removal of the embolus by a competent surgeon may be performed.

Pulmonary emboli may result from thrombi not only in the auricles but also in the veins of the legs and pelvis. Even when heart disease is present, for example, coronary occlusion or heart failure, the legs may

be the source of the pulmonary embolism. Venous thrombosis is common after operation. When it is present in the legs and there is recurrent pulmonary embolism, ligation of the femoral vein may be essential and also the use of heparin has been advocated. It is given as a continuous intravenous saline drip. The usual amount of fluid per day is 2 liters, the dosage of heparin being 30 cc. per liter. Several hours after beginning the injection the clotting time should be ascertained and this should be repeated frequently. The clotting time desired is 15 minutes and the dosage of heparin can be regulated accordingly. If there is heart failure the amount of fluid should be curtailed and the heparin given in 5 percent dextrose solution instead of ordinary saline. An alternative method of administering heparin is the injection given every 3 to 4 hours of 10 to 15 cc. of heparin directly into the vein. The treatment may be maintained for several weeks. The use of heparin has also been advocated to prevent thrombosis after operation and mural thrombosis in coronary occlusion, but its value in these remains to be determined. When thrombophlebitis is present in the legs paravertebral ganglion block has been performed with relief of the phlebitis. It operates by overcoming the spasm in the veins and arteries attending phlebitis.

When a large pulmonary embolus has occurred, chief reliance has been placed upon oxygen. The surgical removal of such an embolism should be reserved for the rare ideal case where immediate diagnosis and operation are possible.

HEMOPTYSIS.—In mitral stenosis this symptom is usually associated with pulmonary infarction. It may also occur in pulmonary edema, which is not common in mitral stenosis. A third form sets in paroxysmally and may be profuse; the explanation of this type is obscure, since there is no evidence of failure in the lungs or systemic system. The treatment is chiefly rest and sedation. Morphine should be given with caution since it may depress the cough and so favor aspiration of blood into lungs.

HYPERTENSION

HYPERTENSIVE ENCEPHALOPATHY.—In the course of essential hypertension, particularly the malignant phase, and in acute nephritis, hypertensive crises may occur during which the patient becomes acutely ill with such symptoms as severe unremitting headache, temporary blindness, paralysis, aphasia, convulsion, the so-called syndrome of hypertensive encephalopathy. It is usually associated with a sudden rise in pressure in the blood and spinal fluid. The attack may be brief, but if it persists relief may be afforded by 20 cc. of a 10-percent solution of magnesium sulphate given intravenously at a slow rate. Occasionally withdrawal of spinal fluid is beneficial and hypertonic dextrose, 50 cc. of a 50-percent solution, has been injected by vein with success.

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PAROXYSMAL HYPERTENSION.—A pheochromocytoma, usually situated in the adrenal medulla, may cause attacks of increased blood pressure associated with symptoms similar to those produced by injection of adrenalin, such as palpitation with marked tachycardia, vertigo, blanching, flushing, etc. The systolic blood pressure may go beyond 200. The diagnosis may be made by x-ray after perirenal insufflation. During the acute episode considerable sedation may be necessary and magnesium sulphate, 20 cc. of a 10-percent solution by vein, but operation usually affords a complete cure.

ANEURYSM.—As a rule a dissecting aneurysm of the aorta forms in association with hypertension. When extensive it may produce a syndrome simulating acute coronary artery occlusion, and rupture may rapidly result in death. The pain in dissecting aneurysm may radiate into the back or lower extremities. In the nonfatal cases the pain may be very severe and require repeated large doses of morphine, 0.03 gram (gr. $\frac{1}{2}$), and the shock may be profound. If the blood pressure is high nitroglycerin, 0.6 milligram (gr. $\frac{1}{100}$) should be given frequently. Occasionally a luetic aneurysm of the artery may rupture. In a case personally observed, the arterial swelling above the clavicle was seen and felt to disappear soon after the onset of shock. This condition is fatal.

CEREBROVASCULAR ACCIDENTS.—Such symptoms as stupor, convulsion, aphasia, hemiplegia, when not due to brain tumor or other local disease, are often the result either of hypertension, with cerebral hemorrhage or perhaps temporary spasm, or of heart disease with cerebral embolism. Among the latter the chief causes are mitral stenosis, particularly with auricular fibrillation, subacute bacterial endocarditis, and myocardial infarction following acute coronary artery occlusion.

The majority of cases survive a first or second cerebral accident. No special treatment is required unless persistent coma develops, in which case aminophyllin, 0.12 grams (gr. 2), should be injected intravenously and repeated if necessary. Lumbar puncture, phlebotomy, magnesium sulphate, 20 cc. of a 10 percent solution by vein, have been tried, but their effect is difficult to evaluate.

HYPOTENSION

SYNCOPE.—Fainting spells are associated in the lay mind with serious heart disease. Actually, syncope is not common in organic heart disease; with the latter it is associated chiefly with heart block and aortic stenosis and occasionally with acute coronary occlusion or paroxysmal tachycardia. As a rule the faint results from a disturbance of the autonomic nervous control of the vascular system, and actually the term "vaso-vagal" attack has been employed; usually the person becomes pale, and the blood pressure and heart rate fall. Also one

should always think of hemorrhage as a cause of syncope. Fainting is apt to attend, in certain persons, some unusual sight or event such as an injection needle, blood, sitting in a dentist chair, rising from the recumbent position, etc. Since syncope is associated with a temporary cerebral anemia, the head should be lowered between the knees. If the person is not revived, atropin sulphate, 0.002 gram (gr. $\frac{1}{50}$), or adrenalin, 0.5 to 1 cc. of 1:1000 solution, should be injected subcutaneously. If the fainting is recurrent, tincture of belladonna, $\frac{1}{2}$ cc. (min. 10), ephedrin sulphate, 0.30 gram (gr. $\frac{1}{2}$), or $\frac{1}{4}$ paradrin, 0.01 gram (gr. $\frac{1}{4}$), should be given three times a day after meals.

One type of syncope has been shown to be due to hypersensitivity of the carotid sinus, a collection of nerves around the bifurcation of the common carotid artery. Carotid sinus sensitivity is often associated with arteriosclerosis but may occur in persons with a normal cardiovascular system. An attack may be induced by any pressure on the sinus, whether by an enlarged gland in the neck or by an external factor or by sudden movement of the neck. A clergyman was recently seen who fainted only on Sundays while delivering the sermon, because on that day he wore a high collar. Carotid sinus sensitivity as a cause of syncope should be confirmed by inducing this symptom by applying pressure on the sinus, first on one side and then on the other while the patient should be sitting. Ephedrin sulphate, 0.03 gram (gr. $\frac{1}{2}$), or paradrin, 0.01 gram (gr. $\frac{1}{4}$), three times a day, or atropin sulphate, 0.0006 gram (gr. $\frac{1}{100}$), may prevent attacks and in a few cases excision of the sinus has given permanent relief.

A not uncommon cause of fainting is postural hypotension, encountered in normal persons and in such conditions as central nervous system cord tumor, tabes, hypertension, neurocirculatory asthenia, etc. When the upright position is assumed by these persons, the normal compensatory splanchnic constriction does not occur, the cardiac output is diminished, and the blood pressure falls. If no cause is found for the condition, atropin, ephedrin and paradrin in the above doses by mouth and an abdominal belt may be tried.

SHOCK.—Peripheral circulatory failure or collapse is characterized by cold, clammy skin, feeble pulse, low blood pressure and collapsed superficial veins. It results from a diminution in the circulating blood volume and venous return to the heart. There are numerous causes of this, such as operation, trauma, hemorrhage, burns, pneumonia, pulmonary embolism, coronary occlusion, ruptured viscus, acute pancreatitis, anaphylactic reactions, Addisonian crises, diabetic acidosis, heat stroke, hypoglycemia.

In addition to tachycardia, embryocardia is often present and gallop rhythm is occasionally heard, especially in coronary occlusion, suggesting myocardial impairment.

If the shock is caused by hemorrhage, an effort should be made to control this rapidly. In injury to an extremity a firm bandage or tourniquet should be used. If the injury is mild, large bandages bound over single layers of wool have been suggested to limit plasma loss. If the arm or leg is very severely injured and cannot be saved, a tourniquet should be applied close to the upper level of the wound. The subject should be kept warm and the foot of the bed elevated. If restlessness is present, morphine, 0.015 gram (gr. $\frac{1}{4}$), should be given and may be repeated. However, not more than 0.03 gram (gr. $\frac{1}{2}$) should be given in 6 hours in order to avoid respiratory depression. If the latter or cyanosis is present oxygen should be used. Digitalis is to be avoided.

The most important principle in treating shock is administration of fluids and salt in order to restore blood volume. When there has been considerable hemorrhage a blood transfusion naturally is the most effective way of administering fluids. Recently the use of blood plasma instead of whole blood has been emphasized in shock not caused by blood loss. It is especially suitable for use in traumatic shock, for example in battle, since it can be kept for long periods and thus is ready for immediate use. Its preparation is simple and it can be sent long distances. At the present time large quantities are being collected in this country and shipped to England. If whole blood or plasma is not available, and in conditions with loss of large amounts of fluid and minerals, isotonic sodium chloride (0.85 percent) with or without dextrose 5 to 10 percent should be administered in the form of an intravenous drip. The usual rate of injection is 40 drops a minute and several liters can be given daily. If the dehydration and vomiting are extreme, the rate should be increased. The amount of fluid and the rapidity of injection must be considered carefully, particularly in the presence of heart disease, lest cardiac failure be precipitated. We have observed several cases of pulmonary edema following a direct transfusion. Acacia, formerly much employed, has not stood the test of time. Similarly it would seem that the common use of 50 percent dextrose solution is unwise, since in shock the tissues are dehydrated to begin with.

In spite of the traditional use of vasoconstrictor and stimulant drugs in the treatment of shock, we believe that they should be avoided except in such conditions as spinal anaesthesia and anaphylactic shock, in which adrenalin, 0.5 to 1.0 cc. of the 1:1000 solution, should be given subcutaneously. In shock small doses of morphine are helpful as outlined above.

Special indications in particular types of shock, such as diabetic acidosis, hypoglycemia, Addisonian crises, will be apparent and are outside the scope of this paper. A word of caution is pertinent, however, concerning the use of pellets of desoxycorticosterone in Addison's

disease. This has sometimes resulted in a rise in blood pressure followed by left heart failure, even pulmonary edema. The dosage must, therefore, be carefully determined. Shock due to heat, as may occur in the boiler room, may be prevented by the ingestion of salt in capsule form.

TRAUMA.—Since the two are very often associated, the treatment of shock is of paramount importance in trauma, whether the latter involves the heart or other parts of the body. The treatment of shock in trauma is the same as that outlined for shock in other conditions.

The heart may be injured directly by penetration of gunshot and shell fragments or such instruments as a knife, sword, or bayonet. In addition, *commotio* or *contusio cordis* may be produced indirectly by a crushing fall or blow against the chest or abdomen. In *commotio cordis* there are no anatomical changes in the heart muscle but a physiological derangement occurs. In contusion of the heart there are areas of hemorrhage and myomalacia. The accident need not be very severe. Thus a young man was struck in the abdomen with a football during a game and collapsed; postmortem examination revealed contusion of the heart.

The diagnosis of injury to the heart in penetrating wounds of the chest is made on the development of pericardial effusion as a result of hemorrhage. The electrocardiogram often helps to make the diagnosis of pericarditis, the rs-r segment being elevated in two or more leads. Although some writers advise immediate operation and repair of the cardiac laceration, we believe that surgical intervention should be withheld unless there is evidence of tamponade of the heart, such as dyspnea, engorged cervical veins, tachycardia and rise in venous pressure. Naturally such a patient must be observed very closely for these signs, and operation performed as soon as they appear. In operations on the heart the local application of metycaine and procaine to the epicardium has been advocated to prevent the onset of and to arrest arrhythmias. Furthermore, it has been advised that if ventricular fibrillation sets in, the heart should be massaged and stimulated with 60 cycle A-C current of 1 ampere passed thru two padded electrodes.

The clinical picture of *commotio* and *contusio cordis* is variable. There may be precordial pain, collapse, gallop rhythm, drop in blood pressure and, especially in *commotio*, arrhythmias and engorgement of the liver, the picture of acute right heart failure. The treatment is essentially the same as in coronary occlusion.

ARTERIO-VEINUS ANEURYSM.—Gunshot wounds involving artery and vein may result in the formation of a communication between these. If there is a considerable arterio-venous shunt the increased venous return to the heart raises the cardiac output and work of the

heart, producing cardiac enlargement and eventually heart failure. It is essential, therefore, to make the diagnosis and ligate the aneurysm early.

OPERATIVE PROCEDURES

The problem of surgical intervention for extracardiac emergencies frequently arises in patients with cardiac disease. Operation should not be performed in the presence of heart failure or after a recent coronary occlusion unless absolutely imperative. If operation is performed in a person with failure, rapid digitalization should be carried out; this may be done intramuscularly, 6 to 8 cat units, 0.6 to 0.8 gram (gr. 9 to 12), being given within 6 to 8 hours, or strophanthin 0.3 milligram (gr. $\frac{1}{200}$) may be injected intravenously. In the presence of severe hypertension, chloral hydrate 0.6 gram (gr. 10) should be given and repeated.

Persons with compensated heart disease tolerate operation well as a rule but occasionally may go into shock or left heart failure. The former may be prevented, when a formidable operation is faced, by slowly administering a small quantity of fluids intravenously during and after the operation. The treatment of shock has been outlined. We have observed several instances of pulmonary edema after operation, particularly in mitral stenosis. It has usually responded rapidly to morphine but the other therapeutic measures in acute left heart failure may be required. If there is any doubt concerning the compensation of the heart prior to operation, digitalization should be carried out.

We believe that in persons with coronary artery disease operation may be a factor in the onset of coronary occlusion, although more commonly of coronary insufficiency. These people, therefore, should be subjected to the simplest type of anesthesia and operation permissible, and particular care observed to avoid anoxemia. It has been shown that the majority of modern anesthetics diminish the oxygen saturation of the blood, some as low as 65 percent. This may be dangerous when coronary sclerosis is present since the coronary circulation is diminished to begin with and further anoxemia may lead to acute coronary insufficiency. Therefore, the patient should not be permitted to become cyanotic; oxygen should be administered early and freely. It is also given if the respirations are shallow or bradycardia sets in. The most desirable anesthetic in patients with heart disease is a local or regional one, since the oxygen saturation is not affected. Also, if cyanosis or shock appears, it is very simple to give oxygen if local anesthesia has been used. If a general anesthetic must be employed, ether should be chosen and administered preferably by the open drop method, or it may be combined with ethylene, which,

it must be remembered, is inflammable. Nitrous oxide is contraindicated, since oxygen cannot be used in adequate concentration with it. Although spinal anesthesia is frequently used, we believe that it is dangerous since it may lower the blood pressure and cause shock. If cyclopropane is used at all, it must be administered cautiously since it increases the irritability of the heart muscle and causes bradycardia and arrhythmias. Avertin is best avoided since it depresses respiration and lowers the blood pressure. Preoperative medication, particularly intravenously, should not be given, since the barbiturates, morphine, etc., lower the blood pressure and oxygen saturation. If they are given, small doses should be used.

ACUTE ANOXEMIA

A number of conditions produce a deficiency of oxygen in the blood and other cardiovascular disturbances resulting in anoxemia of the body tissues, chiefly the brain. They include carbon monoxide poisoning, the inhalation of war gases such as chlorine and phosgene, flying at high altitudes, lack of oxygen in trapped submarines, etc. The generalized anoxemia may induce changes in the heart similar to those already described in coronary insufficiency, that is, disseminated focal areas of necrosis and hemorrhages, especially in the subendocardium and papillary muscles.

The amazing increase in aviation, both in peace and in war, makes the effect of high altitudes upon the body a subject of paramount importance. As the altitude increases the partial pressure of the oxygen in the inspired air decreases, producing a drop in the oxygen saturation of the blood. This oxygen deficiency, as well as the low temperature, at high levels is compensated for by a rise in heart rate and sometimes in blood pressure, resulting in a marked elevation of cardiac output and the work of the heart. Since physical exertion increases the demand for oxygen the flyer finds movement at high altitudes difficult as this further increases the anoxemia. It is for this same reason that sailors trapped in a submarine wait motionless for help.

Another factor in producing anoxemia is centrifugal force, the effect of which is greatest in dive bombing. It results in a pooling of blood in the lower part of the body and in a diminished venous return to the heart and a fall in blood pressure. Blood does not flow to the brain and a "blackout" of that organ may occur.

The effect of altitude sickness may be prevented by the proper use of oxygen and the avoidance of excessive heights. However, prevention begins with the proper selection of candidates for flying. Certain vasomotor types of persons, particularly those with postural hypo-

tension, react unfavorably to moderate altitudes, for example, by fainting. They should not be allowed to ascend above 10,000 feet. Patients with heart disease also should be restricted to this maximum level and particular care should be taken of their oxygen supply. No person with heart disease is permitted to act as pilot. If an ascent to high altitudes is contemplated, suitable clothing for keeping the body warm is worn and the flyer is advised to move about as little as possible and to expend a minimum of energy unless oxygen is readily available.

The symptoms of anoxemia come on insidiously and the senses are dulled to such a degree as to prevent the perception of increasing distress; hence, the importance of using oxygen before the onset of symptoms. Collapse or coma may occur before the flyer realizes that he is suffering from oxygen want. At high altitudes it is essential that, as the oxygen is being drawn into the mouth, the nostrils should be closed. If the oxygen apparatus goes out of order, or in jumping with a parachute, the lungs should be filled with oxygen and the breath held until a descent has been made. For emergency parachute jumps from very high altitudes a special face mask with a small portable tank of oxygen is essential. For flights above 19,000 feet sealed and positive pressure cabins have been used. In order to offset the effects of centrifugal force in dive bombing, the flyer has been advised to assume the crouching position and to use an abdominal belt since these diminish the fall in blood pressure. The use of a high seat has been suggested, necessitating the pilot to lean forward in order to see his objective.

Attention must also be paid to the escape of carbon monoxide from the exhaust while flying. The affinity of this gas for hemoglobin is three hundred times as great as that of oxygen. The combined effect of carbon monoxide and high altitude is particularly harmful as both cause anoxemia. The treatment of carbon-monoxide poisoning consists of administering oxygen combined with 5 to 7 percent carbon dioxide, artificial respiration, and repeated blood transfusions.

WAR GASES

Although war gases such as chlorine and phosgene do not affect the heart directly they may produce sufficient damage to induce pulmonary edema with generalized anoxemia. In such cases it has been found useful to perform phlebotomy, withdrawing about 500 cc. It has also been suggested that full digitalization be carried out rapidly, beginning with 6 to 8 cat units, 0.6 to 0.8 gram (gr. 9 to 12).

NOTE.—I wish to thank my colleagues doctors H. L. Jaffe and S. Dack for their suggestions, criticisms and unremitting help in the preparation of this article.

HYPERSENSITIVITY OF THE CAROTID SINUS

A CASE REPORT

By Lieutenant Commander Julian Love, Medical Corps, United States Navy, and Lieutenant Commander Tilden I. Moe, Medical Corps, United States Navy

Heymans¹ was among the earlier workers to demonstrate the reflex action of the carotid sinus in the regulation of blood pressure and heart rate. Soma Weiss,² however, emphasized the clinical importance of the reflex functions of the carotid bodies in explanation of attacks of sudden and transitory syncope and convulsions which occur in some patients. The value of differentiating the carotid sinus syndrome from epilepsy and narcolepsy is apparent.

The carotid gland and sinus are small structures located in the neck below the angle of the jaw at the bifurcation of the common carotid into its internal and external branches. The physiological function of these small organs is to depress the heart rate and to lower blood pressure. They are connected with the glossopharyngeal nerve which carries impulses to the medulla and cerebral cortex and apparently act through the homolateral vagus.

Weiss and his co-workers³ describe three types of response as a result of stimulation of the hypersensitive carotid gland and sinus. In the first type, there is a fall of blood pressure, slowing of the heart rate to a standstill, and a resultant cerebral anemia with syncope or a Morgagni-Stokes-Adams syndrome. In the second, there is simply lowering of the blood pressure—a depressor effect. In the last, only the cerebral component is affected without circulatory symptoms. If these attacks occur only as a result of mechanical stimulation, the patient is said to have a hypersensitive carotid sinus. The term carotid sinus syndrome is applied to those patients who suffer these attacks spontaneously. The efficacy of carotid sinus pressure in aborting attacks of auricular paroxysmal tachycardia is well known.

CASE REPORT

A. V., Chief machinist's mate, age 40, and of Italian extraction was admitted to the Naval Hospital, Brooklyn, N. Y., complaining of paroxysms of abdominal pain and vomiting. These complaints had been present on and off for about 1 year and had been of increasing severity. Only recently had he noted in addition, some tenderness along the right sternocleidomastoid muscle. The patient later volunteered the information that he had noted faintness and dizziness when his neck was being shaved in the barber chair. There was nothing else in his past or family history which appeared significant. He had never had any venereal disease.

¹ Heymans, C.: Reflexogenic functions of aorta and of carotid sinus. *Compt. rend Soc. de biol.*, 197: 1293, August 1931.

² Weiss, Soma, and Baker, J. P.: Carotid sinus reflex in health and disease; its role in causation of fainting and convulsions. *Medicine*, 12: 297, September 1933.

³ Weiss, Soma, Capps, R. B., Ferris, E. B. Jr., and Munro, D.: Syncope and convulsions due to hyperactive carotid sinus reflex. *Arch. Int. Med.* 58: 407, September 1936.

On examination, a slender somewhat swarthy white male of Italian extraction and 40 years old was noted. He was not acutely ill except when seen during acute attacks of abdominal pain which seemed to be along the lower right costal margin where there was tenderness. He had a small epigastric hernia and three small lipomata in the left abdominal wall and back. A diagnosis of chronic cholecystitis with probable stone was made. Repeated Graham-Cole tests failed to visualize the gall bladder. A subsequent cholecystostomy with removal of gall stones relieved the abdominal complaints, but had no bearing on the condition to be described.

On admission examination, a small nodule was noted along the mesial border of the right sterno mastoid muscle. On palpation of this nodule the patient broke out into a thin sweat, turned pale, complained of dizziness and that things were turning black. He felt as though he were going to faint. When the palpation was over, he made an immediate recovery. On repetition, it was noted that the pulse slowed and stopped and the heart sounds disappeared. Pressure on the left caused only bradycardia.

Laboratory studies indicated nothing of value upon this condition. The blood Kahn was negative. A chest x-ray revealed no significant abnormalities of the heart or lungs.

The electrocardiographic findings were particularly interesting. In figure 13 is shown a section of lead 1 which was taken while the patient was lying normally. Figure 14 shows the effect of mechanical or pressure stimulation of the left carotid sinus. A short period of ventricular arrest, about 3 seconds, was produced, but the auricles continued to beat though at a much slower rate than normal. There was also A-V delay and ventricular or nodal escape. In figure 15, the effects of pressure upon the small tumor over the right carotid gland is demonstrated. There resulted a period of cardiac asystole for over 8 seconds. The patient perspired and became pale, dizzy and unsteady. He then became unconscious and had a short convulsion. Immediate recovery followed release of the pressure. This is shown in figure 16, which is a direct continuation of figure 15.

DISCUSSION

The left vagus nerve endings predominately control the A-V node of Tawara. Pressure upon the left carotid sinus is more apt to effect A-V conduction or cause ventricular standstill than right carotid sinus pressure. The nerve endings of the right vagus terminate, for the most part, at the sino-auricular node and complete cardiac asystole due to arrest of the pace maker results. If this be prolonged, then secondarily cerebral anemia will occur and result in the Morgagni-Stokes-Adams syndrome. However, this results also from the additional mechanism of stimulation of the glossopharyngeal endings which return these impulses cerebrally. Purks⁴ in a series of cases found that stimulation of the right carotid sinus affects the pace maker while left carotid sinus stimulation influences the A-V node.

The apparent increase in the occurrence of carotid sinus syndrome has been demonstrated by Soma Weiss. He believes the syndrome to be the explanation of some knockout blows in boxing and is very

⁴ Purks, W. K.: Electrocardiographic findings following carotid sinus stimulation. *Ann. Int. Med.* 13: 270, August 1939.

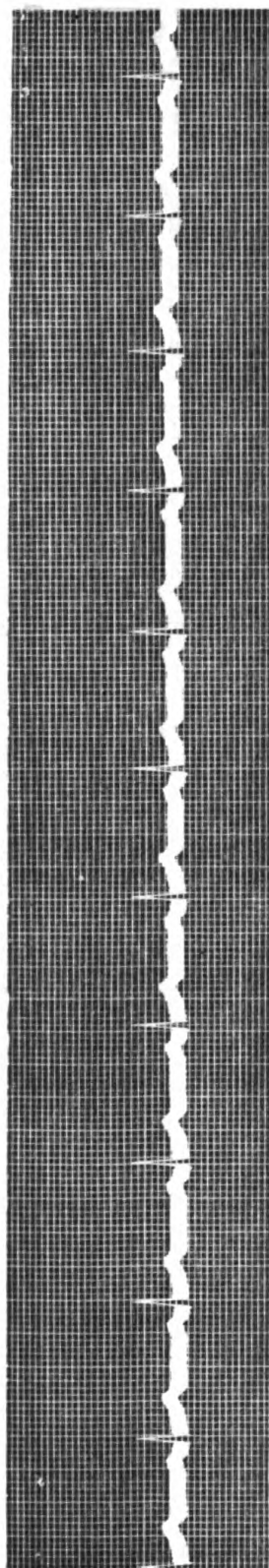


FIGURE 13.—Lead 1 showing normal cardiac mechanism of patient.

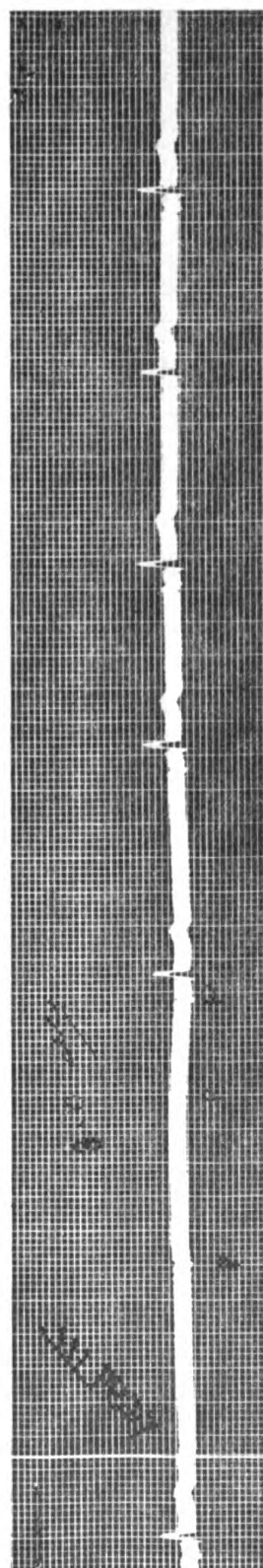


FIGURE 14.—Effect of pressure on left carotid sinus. Note auricular bradycardia and short period (3 seconds) of ventricular asystole. Ordinate at left indicates moment pressure applied.

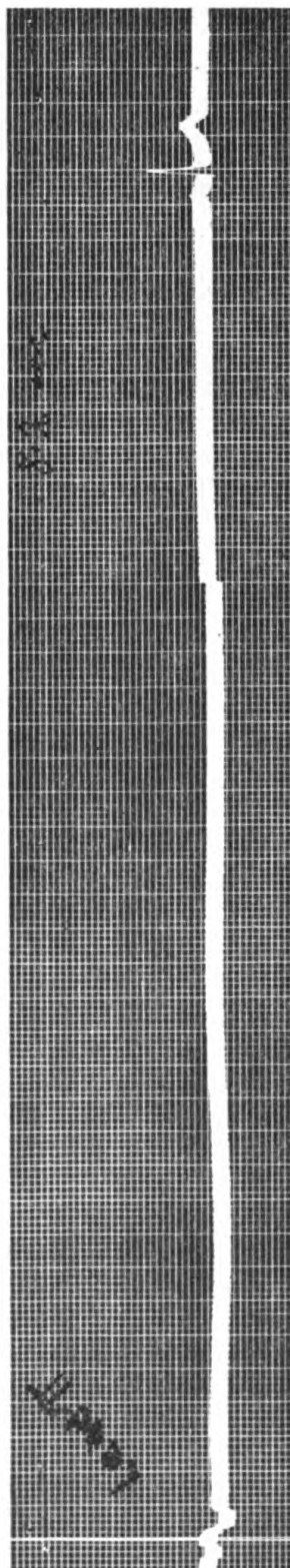


FIGURE 15.—Effect of pressure on right carotid sinus showing asystole for 8.2 seconds.

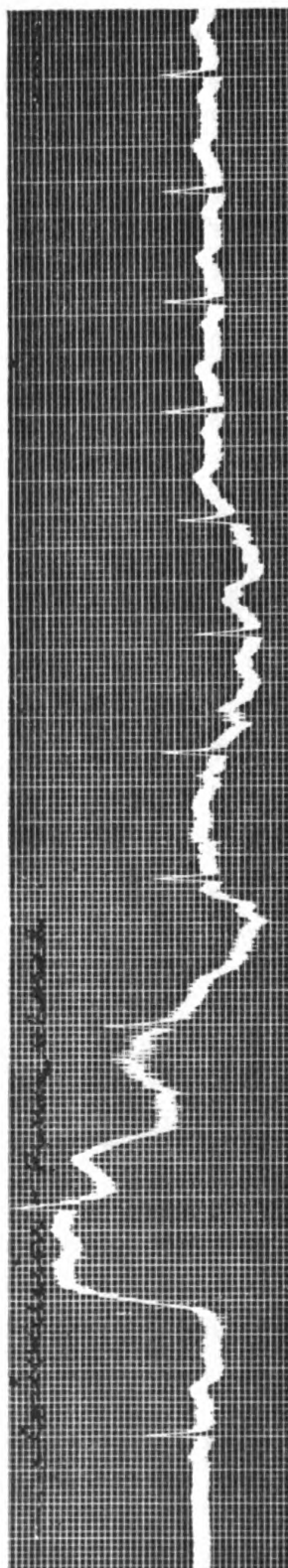


FIGURE 16.—A direct continuation of figure 3 showing the convulsion and immediate recovery from the mechanically induced Morgagni-Stokes-Adams syndrome.

likely the basis of the "glass jaw." Smith of the Mayo clinic⁵ has reported 85 cases.

The treatment of this condition is first a complete explanation of the mechanism to the patient. Secondly, where possible, removal or avoidance of the cause, *i. e.*, removal of external pressure. In those who have spontaneous attacks operative measures aimed at relieving pressure from local growths or abnormalities may be indicated. And as a last resort in severe cases, removal of the gland. A new drug of the sympatheticomimetic series, paredrine, has been synthesized and Nathanson⁶ has reported that it very definitely lessens the sensitivity of the carotid sinus and may be of value in certain indicated cases. In the case of the patient reported above, it was felt that avoidance of local pressure such as tight collars, of skylarking, boxing or wrestling whereby someone might press upon his neck was sufficient since up to now he has never had a spontaneous attack.

Atropine, ephedrine, and benzedrine have also been employed. Procain by local injection will prevent the reflex for a short period.

SUMMARY

1. A patient with a hypersensitive carotid gland and sinus has been reported.
2. Electrocardiographic illustrations of the effects of mechanical pressure on the right and left carotid structures have been submitted.
3. A short discussion of the history, physiologic mechanism, and treatment of the condition has been appended.

AURICULAR FIBRILLATION

By Commander E. C. Carr, Medical Corps, United States Navy

Fibrillation is a term used to express a state of activity in which the muscle fibers or group of fibers in the auricular walls contract without coordination with other fibers or groups of fibers and thus cease to respond in orderly sequence to stimuli arising in the sino-auricular node. There is, therefore, a loss of orderly auricular contraction and ability to send regular impulses to the auriculo-ventricular node and onward to the ventricles. The ventricles respond in an irregular manner both as to rhythm and force and the pulse is irregular as to time, pressure, and volume.

The exact etiology of this condition is unknown. The predisposing etiological factors have been classified as (a) rheumatic, (b) arteriosclerotic, (c) coronary, and (d) miscellaneous, including thyrotoxic

⁵ Smith, H. L.: Fainting attacks resulting from hypersensitive carotid sinus reflexes. *Amer. Heart Journ.* 14: 614, November 1937.

⁶ Nathanson, M. H.: Action of Parahydroxyphenylisopropylamine (paredrine) on the heart. *Ann. Int. Med.* 12: 1355, May 1939.

and syphilitic. It is questionable if syphilis, *per se*, ever causes auricular fibrillation and thyrotoxic cases failing to respond to medication seldom do so due to thyroidectomy alone.

As yet there have been no typical pathological findings that can be classified as characteristic of auricular fibrillation. As the mechanism of production is one of abnormal conduction in the absence of detectable pathological changes, faulty auricular muscular metabolism may be implied. Paroxysmal cases prove that this abnormality is not necessarily permanent.

We may make the assertion that a normal heart does not fibrillate; nevertheless, fibrillators suddenly die from apparent complete heart block without any demonstrable cardiac pathology. The question arises as to whether or not fibrillation increases the strain on cardiac musculature. Lewis states:

The ceaseless activity of the muscle cells in fibrillation should be far more exhausting than the normal activity, with its short periods of activity and its relatively long periods of rest.

Under this hypothesis, fibrillation must be considered as favoring cardiac decompensation. On the other hand, Hyman and Parsonnet state:

No satisfactory explanation has yet been offered to account for the more or less protective mechanism in patients who are suffering from the various types of auricular fibrillation. Indeed, the development of auricular fibrillation even after a coronary attack, seems to offer a better prognostic outlook than any of the other cardiac disturbances that may follow in the wake of the seizure.

Vaquez' simile of a normally contracting heart resembling the rhythmic steps of soldiers crossing a bridge is well known. They break step before crossing and by the same token he considers the rhythmic throbbing of a structurally weakened heart more dangerous than the irregular beating resulting from the onset of auricular fibrillation.

The symptoms may range from slight discomfort only, due to heart consciousness from the irregularity, to complete decompensation and unconsciousness. One patient may complain much of the irregularity and associated symptoms, while another complains but little, except upon physical exertion or mental excitation. In cases of early coronary breakdown, auricular fibrillation may occur and is usually of the paroxysmal type.

Albert S. Hyman and Aaron E. Parsonnet state that in their experience the paroxysmal forms of auricular fibrillation are apt to be of the extremely rapid type with ventricular rates running as high as 200 to 220 per minute. This has not been the writer's experience. However, the onset of rapid fibrillation enhances the distress of the patient. He becomes more dyspneic, cyanosis is inevitably present, and signs of peripheral congestive failure are pronounced. Pulmonary

edema may rapidly ensue and the patient may die very shortly after the onset of these symptoms. It is believed that most of the rapid paroxysmal fibrillators do not live to become permanent fibrillators and, therefore, we see more rapid paroxysmal and few rapid permanent cases.

The attacks may be ushered in by only moderately severe precordial pain or be even unnoticed except upon exertion. However, unless absolute rest is enforced, decompensation may suddenly appear and the patient lose consciousness. No ambulatory treatment should be instituted until the seriousness of the cardiac involvement is determined, for while the cardiac reserve may be compatible with many years of moderated activity, sudden complete block or rapid decompensation and death may occur.

Frederick A. Willus of the section of cardiology of the Mayo Clinic states that he has never observed a case in which purely neurogenic influences caused auricular fibrillation. There must naturally be pathological changes within, or connected with, the inhibitor of accelerator nerves of the heart. However, psychic stimuli will initiate auricular fibrillation in such a heart. Numerous cases could be produced, but one of recent occurrence at a naval hospital will illustrate this fact:

Mr. G. gave a history of previous attacks of cardiac irregularity associated with precordial distress and slight dyspnoea. During these attacks exertion would markedly increase his subjective symptoms. He had suffered approximately 5 such attacks in 2 years prior to admission to that hospital. Shortly after Mr. G. returned to the ward from the operating room, where he had undergone a cholecystectomy, he overheard a patient lying next to him remark to a visitor that Mr. G. was very ill and was going to die. Mr. G. was so upset by this remark that shortly thereafter he began to fibrillate. The writer was called in consultation and assured him that his cardiac irregularity would stop as suddenly as it had started provided he could sublimate his worry concerning it and relax. He was removed to a quiet room and in 48 hours the auricular fibrillation had stopped. A month later after a rapid ride on a hand-propelled laundry truck, he became frightened and again started to fibrillate. This attack lasted 12 hours and stopped without medication.

Many cases have been seen in which the attack was initiated by a thrilling motion picture, prize fight, the witnessing of an accident, or other exciting event. It must, therefore, be held that the exciting factor in some cases of paroxysmal fibrillation may depend upon some neurogenic influence, the "trigger action" of which interrupts the normal excitation wave to produce the "circus movement." In this connection, an interesting case was seen in which the paroxysms were frequently produced by drinking large quantities of ice water on an empty stomach, or by overloading the stomach with a heavy meal. How suddenly to stop these paroxysms remains undetermined.

The diagnosis of auricular fibrillation should cause no difficulty even without the assistance of the electrocardiograph, for there is a marked

discrepancy between the radial pulse and the apex beat. Many of the ventricular contractions are so weak that they do not open the aortic valves and therefore there is no impulse transmitted to the radial pulse. This is known as the "pulse deficit" and, therefore, the pulse is irregular as to volume, time, and force. The electrocardiogram, with its many base lines formed by multiple "P" waves, spoken of in this condition as "F" waves, represented by irregularly spaced and varying amplitude of the "Q-R-S" complex, makes the diagnosis positive.

The prognosis depends upon the existing cardiac pathology. Auricular fibrillation, *per se*, in the presence of minimal ventricular muscular involvement, is compatible with many years of life.

Stroud, Laplace and Reisinger, of the Graduate School of Medicine of the University of Pennsylvania, state:

Of the rheumatic group, those developing their etiological factor before the age of 20 years and beginning to fibrillate between the ages of 20 and 40, the average duration of life was 3 years. Those in this group beginning to fibrillate before 20, their duration of life was less than 1 year. Those in this group beginning to fibrillate after 40 years of age, their duration of life was 5 years. With simple arthritis as an etiological factor, the prognosis as to duration of life seems to be the best, with an average of 2 to 3½ years, and a maximum of 16 years. Of those who acquire their infection before 25 and who are going to develop auricular fibrillation, 86 percent will develop this arrhythmia before 40 and 88 percent will die before 52 years of age.

It must be remembered that the extent of myocardial damage is not indicated by the onset of auricular fibrillation, nor is the development of auricular fibrillation an indication of the amount of myocardial damage. The amount of cardiac reserve is the all-important prognostic indicator.

The treatment depends upon the amount of cardiac embarrassment, decompensation, and subjective symptoms. The subjective symptoms may be so mild that no therapeutic regime is indicated. The writer has seen patients whom the attending physician has had in bed for weeks with the only subjective symptoms being the slight cardiac irregularity unassociated with signs of decompensation, even upon exertion.

There are those who are still over-enthusiastic concerning the effects of quinidine, but the writer believes this drug should not be used empirically in all cases of auricular fibrillation. All fibrillators are paroxysmal before they become permanent. These paroxysmal attacks vary from minutes to days with frequent restoration to normal rhythm regardless of treatment and in the medication of these cases, false therapeutic effects may frequently be the conclusion.

A statement by Stroud, Laplace, and Reisinger may illustrate a waning faith in drug quinidine therapy:

In a previous paper we report our enthusiasm at these (quinidine) results. Since then, however, having followed these cases in the interim, our views have become less sanguine. These are mostly well selected cases with the most favor-

able prognosis and with a minimum of determinable myocardial damage * * * nevertheless their average duration of life following the onset of auricular fibrillation was only slightly over a year longer than the patients who were considered unsatisfactory for quinidine therapy and exactly the same as that of patients who had received quinidine without the restoration of normal sinus rhythm.

This is in accord with recent observations of White and Wolff, of Boston, who state:

There is no definite evidence that it (quinidine) prolongs life or reduces mortality.

In the majority of the writer's cases he has been able to obtain a history of many months of normal rhythm intervening between mild transient attacks of fibrillation early in the life of permanent fibrillators. Drugs may therefore easily be given unwarranted credit for maintaining a normal rhythm during the early life of a fibrillator, when even without drug therapy fibrillation might not have occurred. Many patients under the influence of quinidine, with or without restoration to normal rhythm, frequently complain, "My heart seems to be in a vise," "It feels as though someone is squeezing my heart," "I feel as though my heart is constricted," and other such references.

Digitalis must be considered the drug of choice in the treatment of this condition and its use should be governed by the signs of decompensation and the patient's subjective symptoms. In the absence of both these conditions no medication is indicated except for the etiological factor, if determined.

Gitalin (amorphous), an active glucoside of digitalis, is an excellent form of digitalis therapy in this condition, due to its concentration, rapid action, and slightly more prolonged effect upon the cardiac musculature.

It is possible that large doses of sodium iodide will prevent paroxysms of auricular fibrillation. The writer has used it with excellent results and it is the only drug in his experience which he feels has prevented attacks of auricular fibrillation. One case to whom it was given had his original attack of fibrillation in 1928. His paroxysms had gradually increased in frequency from approximately one every 2 months, lasting from minutes to a few hours in duration, to paroxysms of daily occurrence in June 1938, lasting for hours and often days in duration. He received sodium iodide in 60-grain doses twice daily for 1 week and thereafter 60 grains daily for 2 weeks, at which time the drug was discontinued. Shortly thereafter he again started to fibrillate. Treatment was resumed and fibrillation was controlled in 3 days. He has continued taking the drug and to date has had no recurrence of his fibrillation.

It is not considered at present that sodium iodide will stop cases of auricular fibrillation, but it is felt that it has possibilities of preventing the recurrence of the paroxysms. It is hoped that others will use this

medication so that its specific effect may be determined in a large series of cases of auricular fibrillation.

SUMMARY

Auricular fibrillation gives no indication as to the degree of myocardial damage.

The presence of auricular fibrillation, *per se*, does not dogmatically indicate the necessity for therapeutic intervention.

The degree of decompensation and subjective symptoms should be the guides to treatment.

Quinidine sulphate is not a specific in the treatment of auricular fibrillation.

Digitalis should be used in the presence of subjective symptoms and signs of failing cardiac reserve.

Sodium iodide is a drug, the effects of which are most encouraging in preventing the recurrence of paroxysmal attacks of auricular fibrillation.

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HYPOTENSION IN AVIATION

WITH A REVIEW OF 159 FATAL CRASHES

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It is well known that the vasomotor system is the most sensitive part of the body; and, while the selection and care of the aviator from the cardiovascular standpoint have proved an interesting subject for investigation and study, recent investigation has shown that more emphasis should be placed on hypotension as a factor in assessing

standards of fitness for aviation training. We have been prone to look upon persistently low blood pressure as entirely compatible with a normal state of well-being and health, but whether it is compatible with the stress and strain incident to the violent flying indulged in by naval aviation, especially in wartime, is another matter.

Aviation within the past few years has undergone such enormous development in the design and construction of airplanes making for increased power, stability, speed, climb, and maneuverability that one might think the human machine—the flier—had been somewhat overlooked. On the contrary, there have been remarkable advances in aviation medicine, the results of which have overwhelmingly shown in a lowering of aviation accidents since the late World War. It is particularly gratifying to note that, despite the increased scope of naval aviation activities in the fleet during the past year, the accident record was 31 percent less than during the fiscal year 1938.

The problems that confront the flight surgeon still are the problems that confronted him 15 years ago, namely, (a) nervous stability or the psychic factor, (b) respiratory, and (c) cardiac efficiency. These are the important factors in the make-up of a naval aviation pilot. The fundamental physiological and psychological consideration of the problems as understood and reported at that time have not materially changed, but a better and more thorough understanding of these conditions has been developed which, it is believed, will be of interest. These problems have been altered somewhat with the increasing power and refinements of aircraft. The psychological aspect is undoubtedly the most important and difficult problem in the selection of a candidate for naval aviation; just what part it plays in hypotension is difficult to say. Improvements in the supply of oxygen for high altitudes have for the present relegated the respiratory factor to the background. With the stress and strain incident to increasing speeds likely to evoke vast forces into action on the bodily mechanisms of the pilot, it has become necessary to put more stress on the study of cardiovascular efficiency. As Marshall ¹ has pointed out, the forward speed that a pilot can endure without discomfort, is far in excess of the speed that any airplane is capable of at present. It is only change of direction, acceleration or deceleration that brings centrifugal forces into play. Furthermore, during these intervals, which may be only a few seconds, the pilot's body mechanism is subjected to these forces which experiments have shown drain the blood from the upper parts of the body. Moreover, from this sudden and rapid change strain is brought to bear in many other ways on the circulatory system during flying. Flack and Bowdler ² stated that from a consideration of blood-pressure records, the conclusion is forced upon us that stress of service in high flying almost invariably produces

¹ Marshall, G. S.: *Jour. Roy. Aero. Science*, 37: 389, 1933.

² Flack, M. and Bowdler, A. P.: *The Medical Problems of Flying*, London, 1920.

a low diastolic pressure, due probably to lessening vasoconstrictor tone of the arterioles.

Whatever the cause, hypotension should always be looked upon with suspicion in selecting candidates for aviation. They might be classed with the "fainters" as potential aviation hazards. However, it is a fact that we have several pilots in naval aviation today with exceptionally low blood pressures, who have been performing their duties in a highly creditable manner while careful and continuous observation has failed to show any objective symptoms to the contrary.

Our present conception of the hypotensives in aviation has been almost exclusively due to Treadgold³ who made 20,000 calculations in the Royal Air Force as a frame for British standards. He noted that estimations of systolic pressures alone may be misleading. Where a pressure of 125/100 would be suggestive of pathology, one of 155/75 in a young adult under excitement of a medical examination is usually of no significance. Opposite interpretation would be placed on such cases in many instances by a medical examiner relying on systolic pressure alone. It would appear, therefore, that diastolic pressures between 70–80 mm. of Hg regardless of the systolic reading could be classed as of no significance in healthy young adults. However, a diastolic reading from 80 to 90 mm. of Hg would be open to question, and anything above 90 mm. of Hg diastolic should seldom be accepted for aviation training without renal investigation.

WHAT IS HYPOTENSION?

Hypotension may be defined as a condition of the circulation in which the systolic or diastolic blood pressure or both falls below a certain figure. Most authorities agree and accept 110 mm. Hg as the lower level of systolic blood pressure. Osler and McCrae⁴ regard anything below 110 mm. Hg as hypotension. Likewise, Cecil⁵ is in agreement with these findings, but neither of these authors defines diastolic standards. Others regard a systolic pressure of 110 mm. Hg and a diastolic pressure of 66 mm. Hg as the lower limit in males, but there is apt to be some variation. MacWilliam^{6,7} considers a systolic pressure below 110 mm. Hg and a diastolic pressure below 66 mm. Hg as definite hypotension; Miller⁸ gives 105 mm. Hg for systolic and 60 mm. Hg for diastolic as bottom levels; Snell⁹ quotes U. S. Army flying standards that systolic pressures less than 105 mm. Hg are disqualifying for flying in all cases. The standard used in the Royal Air Force for some time is to consider any

³ Treadgold, H. A.: *Lancet* 1: 733-740, April 1933.

⁴ Osler, W., and McCrae, T.: *Principles and Practice of Medicine*, 11th ed. D. Appleton-Century Co., New York.

⁵ Cecil, R. L.: *Textbook of Medicine*, 3d ed. W. B. Saunders Co., Phila. 1935.

⁶ MacWilliam, J. A.: *Physiological Review*, 5: 303, July 1925.

⁷ Croll, W. F., Duthie, R. J., and MacWilliam, J. A.: *Lancet*, 1: 194, January 1935.

⁸ Miller, W. H.: *Jour. Avia. Med.* 7:161, December 1936.

⁹ Snell, C. F.: *Jour. Avia. Med.* 7: 12, March 1936.

systolic pressure below 110 mm. Hg and a diastolic below 70 mm. Hg as evidence of hypotension.

We have no established disqualifying standards for hypotensives in the United States Navy. Regulations governing blood pressure in general are quoted as follows: "Systolic blood pressure if the examinee is over 25 years of age, should not persistently exceed 145 mm. Hg; if examinee is 25 years of age or under, systolic pressure should not persistently exceed 135 mm. Hg. The diastolic should be roughly two-thirds of the systolic." From the foregoing it will be observed that we have no definite standard by which we can classify hypotension in aviation as regards the lower limits of systolic and diastolic pressure. In examining candidates for flight training I have qualified several candidates whom other flight surgeons now consider definite hypotensives and questionable aviation material for military purposes.

It would appear, therefore, that most authorities agree 110 mm. Hg systolic and 70 mm. Hg diastolic should be the lower limits. This standard will be used as a basis in this discussion. The exact level of the systolic pressure is not difficult to obtain yet it is apt to fluctuate due to emotional or other causes and is not usually considered as valuable as that of the diastolic pressure which may be more difficult of estimation. Kilgore¹⁰ stated that although the auscultatory method for estimating diastolic blood pressure was the best available, in approximately 10 percent it was impossible to determine it accurately. The difficulty increases, especially with a person with a very low diastolic pressure. The lower the pressure the harder it is to estimate correctly. In fairness and justice to the examinee, one has to record many readings over several days to be absolutely sure of his result.

ESSENTIAL HYPOTENSION

Essential hypotension is a rather unsatisfactory name applied to a symptom complex in certain individuals with a persistently low blood pressure. It may be associated with a group of subjective symptoms such as dull headache, dyspnea on exertion, palpitation, vertigo, easy fatigability, inability to concentrate, and giddiness on change of posture. The condition appears to be rare. Croll and Duthie⁷ could find records of only 18 cases. Other cases have been reported by Alvarez and Roth,¹¹ Chew, Allen and Barker,¹² Davis and Davis,¹³ Korns and Randall,¹⁴ Langston,¹⁵ and Weis.¹⁶

¹⁰ Kilgore, E. S., Berkley, H. K., Rowe, A. H., and Stabler, W. K.: *Arch. Int. Med.* 16: 927, 1915.

¹¹ Alvarez, W. C., and Roth, G.: *Proc. Staff. Meet. Mayo Clin.* 10: 483, July 1935.

¹² Chew, E. M., Allen, E. V., and Barker, N. W.: *Northwest Med.* 25: 297, August 1936.

¹³ Davis, P. L., and Davis, M. S.: *J. A. M. A.* 109: 1247, April 1937.

¹⁴ Korns, H. M., and Randall, W. L.: *Am. Heart Jour.* 13: 114, January 1937.

¹⁵ Langston, W.: *Ann. Int. Med.* 10: 688, November 1936.

¹⁶ Weis, C. R.: *Ann. Int. Med.* 8: 920, February 1935.

The patients usually complain of giddiness on assuming the erect posture and examination has shown that the blood pressure, both systolic and diastolic, falls to very low levels on standing. At times these patients may show pressures within normal limits if taken in the sitting position and this may be misleading. It seems reasonable to infer that between the frank case of essential hypotension and the normal there lies the person whose vasomotor control, although sufficient to prevent symptoms under ordinary conditions, may break down under stress. Such conditions of stress are likely to arise in pilots as a result of lowered oxygen tension, fatigue, and especially centrifugal force while flying. Moreover, the persons most likely to suffer from these effects are those with mild forms of essential hypotension.

The problem of detecting susceptible individuals can be studied by the flight surgeon through two methods suggested by Rook and Dawson¹⁷ (a) a study of the blood pressures of those pilots who have reported symptoms of giddiness or actual syncope while flying or otherwise, and (b) a flying test which subjects persons who appear likely to be affected by the stresses of centrifugal force. However, the effects of flying tests which produce symptoms can usually be overcome by habituation to flying.

FACTORS INFLUENCING HYPOTENSION

Factors influencing or causing hypotension are legion, just as there are considerable differences of opinion as to what constitutes hypotension. Again the fact is often overlooked that the blood pressure of a normal person may fluctuate widely from day to day, and sometimes almost from moment to moment. Some of the factors which have a direct bearing on hypotension may be summed up as follows:

1. It has been observed that in the convalescent or latent stages of certain diseases, faintness or actual fainting in association with a low blood pressure is fairly common, and is apparently due to toxicity. This has probably influenced opinion as to the suitability of hypotensives for flying. It is questionable if this hypotension with syncope attacks is directly due to a lowering of the blood pressure or is simply a manifestation of imperfect vasomotor control.

2. Hypotension in association with tuberculosis is relatively common. Tice¹⁸ states that as a general rule hypotension exists in all stages of tuberculosis. This is confirmed by Cecil⁵ especially in advanced stages with ulcerative lesions. The difference between lying and standing pressure may be greater than normal. Beaven¹⁹

¹⁷ Rook, A. F., and Dawson, D. J.: *Lancet* 2: 1503, December 1938.

¹⁸ Tice, F.: *Practice of Medicine*, 7: 12, 1924.

¹⁹ Beaven, C. L.: *J. Avia. Med.* 5: 34, June 1934.

quotes the cases of 15 U. S. Air Force officers who developed tuberculosis, the average systolic pressure being 106 mm. Hg. However, it appears to be the consensus of opinion among other observers that a low blood pressure is not a reliable sign of tuberculosis.

3. Prolonged mental stress seems to have little effect on blood pressure in healthy adults, the blood pressure varying directly with the pulse rate in these individuals.

4. The endocrine factor has proven to be negligible, except in advanced cases where the endocrine disorder is a disqualification *per se*.

5. Age plays little part as a factor in healthy adults. From 18 to 40 years there is little change in systolic pressure. There may be a rise in diastolic pressure from 18 to 21 years, and a slight fall after 35 years.

6. Body build has little effect if proportionate. The same can be said for weight. Overweight usually carries with it a higher pressure, while hypotension is more common in underweight.

7. Climate affects blood pressure. Roddis and Cooper^{20,21} found the blood pressure lower in the tropics than in temperate zones. This was confirmed by the writer in over 500 examinations done on aviation personnel in the tropics. The British military forces in equatorial Africa showed that hypotension occurred in direct proportion to the heat encountered.

8. Cannon and Mott²² believe that the manifestation of hypotension is the result of fatigue influenced by emotional stress. Based on research and animal experimentation definite changes have been noted in the cortical cells of the suprarenals showing cell membrane rupture, displacement of nuclei, and disappearance of the cytoplasm. The close analogy of the adrenals' influence on the conductivity of brain and liver tissues leads us to believe there is a basis for disfunction of the adrenals caused by psychic conflict. The change in pulse rate and peripheral tone follows as a result of hypotension.

9. Graybiel, Dill, Missiuro, and Edwards²³ found that oxygen lower than 12 percent usually brought about marked blood pressure changes in normal healthy individuals; and, if oxygen tension is gradually lowered, a condition of hypotension supervenes prior to collapse. Similar reactions had been noted several years ago in connection with the rebreather test, which has become more or less obsolete in aviation examinations. Therefore, anoxemia may be a factor in hypotension that requires more investigation.

10. Hypotension is often observed in severe hemorrhage and in shock due to any cause.

²⁰ Roddis, L. H., and Cooper, G. W.: J. A. M. A. 87: 2053, December 1926.

²¹ Roddis, L. H., and Cooper, G. W.: U. S. Nav. Med. Bull. 25: 54, January 1927.

²² Mott, F. W.: War Memoirs and Shell Shock, 1919, New York.

²³ Graybiel, A., Missiuro, V., Dill, D., and Edwards, H.: Jour. Avia. Med. 8: 178, December 1937.

11. Severe anemias and wasting diseases, especially Addison's disease, are frequently attended by hypotension.

12. Myocardial failure may be accompanied by a fall in blood pressure.

13. The emotional component undoubtedly is a factor in hypotension and blood pressure readings in general. This reaction explains at least in part, the low blood pressure readings often found in aviators, who on subsequent examinations show a more nearly basic reading. Athletes who are on a rigid training schedule often show a low average blood pressure. Treadgold's³ group averaged 103/60 mm. Hg. with a pulse of 74. This is in agreement with others. This same investigator also collected a non-athletic group whose average blood pressure was 102/58 mm. Hg. similar to the above athletic group in respect to hypotension but whose average pulse rate was 97. Another symptom noted in the latter group was the evident approach to syncope while blood pressure was being taken. This was evidenced by sweating, pallor, cyanosis and dilation of the pupils—factors which were absent in the athletic group. Therefore, attention should be called to the fact that while low blood pressure may be a bad omen, it can be the acme of perfection. At the same time, working on the hypothesis that blood pressure rises in reaction to an emotional stimulus, and that the rise is in proportion to the degree of stimulation as measured by introspection or some other means, one is bound to be frustrated by these types of individuals. Even the heart rate in a high percent of these hypotensives may have an average rate comparable, at times, to that found in the athletic group. Braun²⁴ suggested that it seems more reasonable to assume that the different cardiovascular patterns noted are quantitative reactions, modified in some cases by physiologic considerations. The athletic group maintain a nearly basic blood pressure and pulse which are supported by an efficient physiologic reserve of power to meet an emergency. This reserve is adequate to meet a harmless emotional situation, such as a physical examination. The athletic type of hypotensive candidate reacts mildly to the emotional stimulus by a rise in systolic blood pressure. The non-athletic and otherwise normal appearing hypotensive candidate lacks the necessary physiologic equipment to carry out the required response, consequently his blood pressure rises then drops with dramatic suddenness leaving only the heart struggling vainly with a thready pulse to maintain the defense of the organism.

INCIDENCE OF HYPOTENSION

The incidence of hypotension has been rather difficult to estimate. It varies as widely with different authorities as does the estimations of blood pressure readings. Treadgold³ found 3 percent hypoten-

²⁴ Braun, L.: *Herz und Psyche in ihren Wirkungen Aufeinander*, Leipzig, Franz decticke, 1920.

sives among fit pilots serving with the Royal Air Force from 1927 to 1931 inclusive. Friedlander²⁵ quoting from various observers noted that the percentage varied from 2.2 to 4.5 in over 9,000 young adults. Chamberlain²⁶ reported that in 60 medical students and doctors there were 15 with a systolic pressure below 110 mm. Hg.; in 4 of these the diastolic pressure was below 70 mm. Hg.; 2 others had a low diastolic with a normal systolic pressure. Therefore, 17 (28 percent) of the 60 cases were hypotensives. Bramwell and Ellis²⁷ examined the circulatory systems of Marathon runners and found the diastolic pressure below 70 mm. Hg. in 4 out of the 28 runners. One man with a pressure of 105/70 was third in the race. Rook and Dawson¹⁸ examined the records of 1,000 consecutive candidates for military service between the ages of 18 to 25 years, and they found an incidence of 12½ percent. In most instances hypotension resulted from low diastolic pressures. Furthermore, it was persistent in less than half the hypotensives who were reexamined once. From the foregoing there seem to be wide discrepancies in the figures on the incidence of hypotension.

In the examination of 716 candidates from civilian life for naval cadets for aviation training at Pensacola, Florida, from 1934 to 1937, I found the incidence of hypotension averaged approximately 9 percent, using as a standard a systolic pressure below 110 and a diastolic pressure below 70 mm. Hg. These candidates were all college students of at least 2 years' training. Their ages ranged from 18 to 28 years. All denied a history of syncope, dizziness, or similar manifestations. On closer questioning, however, 2 percent admitted fainting on one or more occasions during their lifetime but stated "it didn't amount to anything." Forty-three candidates whose pulse rate averaged approximately 100 and blood pressure 103/60 showed evidence of syncope while blood pressure was being calculated, as evidenced by sweating, pallor, cyanosis, especially of the extremities, and dilation of pupils. Seven other candidates fainted, losing consciousness for a minute or more while their blood pressure was being taken. No accurate blood pressure readings were obtained in these cases during the fainting episode. It was noted, however, that the blood pressure was below 100 systolic when the individuals fainted. However, subsequent examination showed these individuals to be hypotensives. The examination was discontinued at this point and the candidate disqualified for flight training. It was particularly noticeable that the above 43 candidates, on being questioned, admitted that they "couldn't stand the sight of blood." They further admitted that they couldn't tolerate pain without feeling nauseated

²⁵ Friedlander, A.: Hypotension, London 1927.

²⁶ Chamberlain, E. N.: *Liverpool Med. Jour.*, 3:235, 1930.

²⁷ Bramwell, C. and Ellis, R.: *Quar. J. Med.* 24:329, April 1931.

and even vomiting at times. Some complained that the sphygmomanometer cuff hurt their arms while taking their blood pressures or that they had pain and tingling in their arm and hand. Changing the cuff or adjusting it or even placing it on the other arm had little effect. They often exhibited considerable apprehension over a cyanosis that occurred below the cuff. None of these symptoms occurred in any of the individuals with a normal blood pressure.

Further observation revealed that the hypotensives were underweight with the exception of three cases whose history and general appearance suggested hypothyroidism. Hypotension appears to be more common in young male adults than is usually imagined. While occasional hypotension is relatively common, many examinations may be necessary to definitely establish persistent hypotension.

RELATION OF HYPOTENSION TO CENTRIFUGAL FORCE

As has been stated previously, any known speed can be borne not only in safety, but also in comfort, provided only, that protection is afforded against the wind. The rapid acceleration or deceleration to or from such speed, however, is another matter, as are the effects of centrifugal force incident to sharp turns at high speeds. In pulling sharply out of a dive, in violent acrobatics or doing a steep turn, forces many times that of gravity may be encountered. The direction in which the force is applied naturally depends on the position of the body in relation to the center around which the turn is effected. These forces are usually measured in terms of "G"—the acceleration due to gravity. The force resulting from any given turn or similar maneuver varies directly with the square of the speed of the plane and inversely with the radius of the circle through which the maneuver is effected. For example, a plane doing a steep turn with a radius of rotation of 300 yards at a speed of 200 miles per hour causes a force of about $3.2+G$; at 300 miles per hour with the same radius the force is more than doubled. If, however, the radius of the circle is increased to 800 yards with a speed of 300 miles per hour the resultant centrifugal force is less than $3+G$. Let us say then that the effective weight of a 150 pound pilot under such circumstances may reach 750 pounds or more. Blood may be forced out of the brain to cause momentary unconsciousness or a "black out" commonly experienced in acrobatic flying and dive bombing. Granting that "black outs" are usually momentary, they can well be fatal under combat conditions with an enemy airman pouring lead or explosive shells at a blind or unconscious pilot. If enough violent flying is indulged in, permanent damage can be done to the central nervous system and the internal organism, as was witnessed a few years ago aboard one of our larger aircraft carriers, when two fatalities occurred within a few hours after carrying out an extensive dive bombing exercise. Actual fractures are not

impossible. If and when speeds of 500 and even 600 miles per hour, which designers are talking about, are reached, the beating the human body will take is obvious. Today's top speeds in level flight in still air are not much above 400 miles per hour.

A particularly disastrous group of fatal accidents occurred in the years 1937 and 1938. Some planes actually disappeared without even a trace. What has been the cause of these unexplained crashes? With the present trend toward perfection in mechanical construction and design, it doesn't seem possible that all these fatal crashes can be explained on failure of matériel alone. Certain it is, one has to consider the personnel factor. In fact, in a large percent of these fatal crashes, pilot error was officially given as the primary cause, where subsequent investigation could be carried out; but what about the planes that disappeared without leaving any trace or sign in average or better flying conditions? Was the pilot careless or grossly incompetent? Such I do not believe to be the case in our naval trained pilot. Could not hypotension be a factor in some of these unexplained crashes? I have in mind 5 cadets that I qualified for aviation training, who were all hypotensives, measured by our standards of today. Their average blood pressure, from many examinations, was 105/66. I followed them carefully through primary training and into advanced training. Three of them developed aeroneurosis and air sickness and never completed primary training; one realized his inaptitude and quit the course during the advanced acrobatic training; the other was killed in the advanced course of training while practicing dive bombing. None of them would admit any symptoms of hypotension other than a feeling of insecurity in the air. Aviators are not exempt from the development of such disorders. In fact, the special hazards of their profession give them greater cause to develop them. Furthermore, it is only because we are dealing with a superior group that we do not find a much higher incidence of these disorders. Probably, if more care were used in assessing flying fitness in regard to hypotensives, these disorders and accidents could be further lessened.

HYPOTENSION IN FATAL AVIATION CRASHES

The present European War, with its high casualty rate from what is known in broad terms as pilot failure, is again raising the question as to whether the human mind and body are being taxed beyond their capacity by the speed and complexity of today's airplanes. Reports from abroad indicate that, at a minimum, for every four pilots killed as a direct result of combat, six lose their lives because their judgment becomes faulty or their bodies are unable to stand the strain. Some American authorities insist that pilot failure is accounting for six out of seven deaths in the war. These casualties would include the men officially entered on the records as having been killed in combat. In

a good many of these cases it is believed that the pilot was shot down by an enemy because he "went to pieces" and presented himself as a perfect target for his opponent's guns. While we are endeavoring to select the best physical and mental specimens as candidates for aviation pilot training, yet a number of these young men are getting killed in peacetime accidents. Might not cardiovascular instability or hypotension be a cause? Again, there is the question whether the human machine is being overloaded due to the complexity of controls of aircraft, especially in switching from contact to instrument flying in adverse conditions.

With a view of finding some possible association between hypotension and fatal crashes, if any, a study was made of the records of 114 naval aviators who were involved in fatal crashes (see table 1). For comparison, a study was made of the personnel of 45 fatal crashes which occurred over the same period in connection with student naval aviation training at a naval air station, also shown in the table.

TABLE 1.—*Personnel analysis of fatal crashes*

	Experi- enced naval aviators	Percent	Student naval aviators	Percent	Total	Total percent
Number of personnel.....	114		45		159	
Average age of personnel.....	29		25			
Body build:						
Height.....	69½		70			
Weight.....	164		154			
Average pulse rate of those with normal blood pressure:						
Prone.....	73		71			
Standing.....	82		79			
Average pulse rate of those with hypotension:						
Prone.....	72		72			
Standing.....	86		84			
Number with normal blood pressure.....	66		20			
Hypotensives:						
Number with low systolic only.....	11		2			
Number with low diastolic only.....	19		19			
Number with both low systolic and diastolic.....	18		4			
Total hypotensives.....	48	42.1	25	55.5	73	45.9
Total crashes due to matériel failure.....	35	30.7	8	17.7	43	27.0
Total crashes due to personnel (pilot error).....	79	69.3	37	82.2	116	73.0
Number of hypotensives in crashes due to matériel failure.....	8	7.0	3	6.6	11	6.9
Number of hypotensives involved in crashes due to personnel (pilot error).....	¹ 40	35.1	² 22	48.8	62	39.0
Number with normal blood pressure involved in crashes due to per- sonnel or pilot error.....	³ 28	14.5	⁴ 9	20.0	37	23.9

¹ 83.3% of all hypotensives.

² 88% of all hypotensives.

³ 42.4% of those with normal blood pressure.

⁴ 45% of those with normal blood pressure.

This table shows the average age, average height, average weight, and average pulse rate both prone and standing, in addition to blood pressure findings. Hypotension is considered here where the systolic pressure is below 110 mm. Hg. and the diastolic below 70 mm. Hg.

In calculating hypotension an estimation of both systolic and diastolic pressures was done. Of the 114 pilots, 48 (42.1 percent) showed some form of hypotension. Studying these 48 cases further as to the cause of fatal crashes from the standpoint of matériel failure or personnel (pilot error), it was found from official reports that 40 (83.3 percent) of the 48 cases were due to personnel or pilot error. Comparing the remaining 66 cases which showed a normal blood pressure, it was found that in 28 (42.4 percent) the cause of the crash was reported officially as due to personnel or pilot error.

Turning now to the table where a study was made of the students under instruction, at a naval air station it will be noted that out of the 45 case records studied, 25 (55.6 percent) showed some form of hypotension. Two showed a low systolic pressure with a normal diastolic, 19 showed a low diastolic only, and 4 had both low systolic and diastolic. From these 25 hypotensives, 22 (88 percent) showed that the official cause of their crashes was due to personnel or pilot error. In the remaining 20 cases with normal blood pressure findings, there were 9 (45 percent) whose official report showed the cause of the crash to be due to pilot error.

Certain relationships, as far as hypotension is concerned, may prove to be a problem worth considering in some of these fatal crashes. It would seem therefore, from the above study, that hypotension points to a factor in fatal crashes due to pilot error.

The relationship of fatal crashes due to matériel failure and those due to pilot error is interesting. The ratio is about 1 : 3. Another factor noted in this series of cases was the higher average pulse rate, particularly the standing rate, among the hypotensives. This is in agreement with Treadgold's³ observation in his so-called non-athletic group which he found to be poor aviation material. The average prone pulse rate for those with normal pressures averages about the same as that noted in the hypotensives, while the standing pulse rate in the hypotensives averages four beats in the seasoned pilots and five beats in the student pilots higher than those with normal blood pressures. This shows that the heart action in hypotensives is apparently more sensitive to mild exertion than in those with the normal blood pressure, or else it is a manifestation of imperfect vasomotor control. It may be that these increased pulse rates are quantitative reactions modified by physiologic factors; that those with normal blood pressures have a better physiologic reserve of power to meet the average emergency without completely "blowing up," as it were, over trivial matters. Every flight surgeon has experienced this phenomena in

the examining room. It appears that the hypotensive endeavors to react properly, but fails through lack of physiologic reserve; the blood pressure then falls and a concomitant increase in the heart rate occurs which is necessary to maintain the defense of the individual. He lacks the physiological equipment to carry out the required response or to react rapidly to stress.

It is realized that the number of cases studied here is too small to draw any but the most tentative conclusions. It is difficult to lay down in concrete terms the course to follow. That comes with a background of experience and further experimentation. There is no doubt that the emotional side plays an important part in cardiovascular stability. Then there is the fatigue factor. Psychological fitness is as important as physical fitness. No one factor alone can supply the answer to the problem of vasomotor or cardiovascular efficiency. All these various factors and combinations must be taken into account in estimating fitness for flight duty.

In the light of the present European conflict and the part aviation is playing and the prominent place it will occupy in the future as an arm of our national defense, more study should be done on the significance of hypotension in selecting personnel for flying and in analysis of crashes.

SUMMARY

1. Hypotension is a rather relative term inasmuch as it has been more or less ill-defined. However, it may be said to exist when the systolic pressure is below 110 mm. Hg., or the diastolic below 70 mm. Hg.

2. Hypotension may be compatible with a normal state of well-being and health, but, if persistent, careful study and caution should be exercised in qualifying these individuals for aviation training.

3. Hypotension may be associated with a momentary dizziness and a fall in the blood pressure to very low levels on assuming the erect posture. This should be a permanent disqualification for flying.

4. Persistent hypotension is not common and its incidence is difficult to estimate. It may not be permanent and it may be influenced by insidious disease or pathology. Occasional hypotension seems quite common in young individuals.

5. In 159 fatal crashes, 73 pilots (45.9 percent) manifested some form of hypotension. Of these 73 hypotensives, in 62 (85.6 percent) the official cause of fatal crash was stated to be due to error of the pilot.

6. Another noteworthy factor in this series of cases was the higher average pulse rate, particularly the standing rate, among hypotensives.

7. Ability of hypotensives to withstand centrifugal force depends upon the reaction of the vasomotor system, modified in some cases by

an efficient physiologic reserve to maintain a nearly basic blood pressure and pulse to meet an emergency, be that a serious or harmless emotional situation. Furthermore, if continuous violent flying is indulged in over a long period of time this reserve can become exhausted due to fatigue and bring about a symptom complex similar to that seen in moderate and severe grades of hypotension with postural giddiness due probably to lessening of vasoconstrictor tone of the arterioles.

8. It seems reasonable, therefore, to conclude that the individual with a normal blood pressure would be better able to withstand continuous violent flying under combat condition over a longer period of time with less exhaustion and fatigue than the hypotensive. In other words he would be less apt to "go to pieces" or commit pilot error under stress.

9. From this study of 159 fatal crashes it appears that more attention should be given to hypotension and its significance in the selection of flying personnel as a further means of lowering crash hazards. If hypotension is present and the individual concerned is unable to increase the diastolic pressure above 70 mm. Hg. on standing, this should be taken as an unfavorable sign for aviation. A fall of, or only a meager rise of blood pressure on change from a prone to standing position shows vascular instability which should be carefully studied in selecting a pilot for military aviation.

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INTRAVENOUS PENTOTHAL SODIUM ANESTHESIA^{1 2}

WITH ILLUSTRATIVE CASES

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INTRODUCTION

Intravenous injection of various agents for the production of anesthesia has been practiced for approximately 100 years, although only recently has this form of anesthesia received wide acceptance. Anesthesia was produced in 1847 by the intravenous injection of ether in normal saline solution by Pirogoff and von Flour.³ In 1872, Ore induced anesthesia by the intravenous injection of chloral hydrate. Both drugs were discontinued for intravenous anesthesia because of

¹ From the Department of Surgery, U. S. Naval Hospital, Charleston, S. C.

² Read before the Medical Society of South Carolina on Feb. 13, 1940.

³ Herb, I. C.: General Anesthesia. Dean Lewis' Practice of Surgery, 1: 40-43. W. F. Prior Co., Inc., Hagerstown, Md., publishers.

high mortality. The early literature contains few articles on intravenous anesthesia, and little progress in the technic of administration was seen until 1911 when Burkhardt developed and discussed his isopral-ether narcosis.⁴ In 1923 Graef reported a large series of cases of intravenous narcosis by the use of isopral-ether mixture, and although it was not considered dangerous from the standpoint of undesirable after effects, it fell into disuse apparently because of the somewhat elaborate mode of preparation and slight increased tendency to hemorrhage.⁵

In 1921 Kirschner made public his use of intravenous injection of avertin as an introduction to ether anesthesia or for operations of short duration. With the gain in popularity of rectal administration of avertin, the intravenous employment of this agent rapidly declined. It appears that the danger of venous thrombosis is the greatest objection to the intravenous use of avertin.^{6 7}

During the past 11 years, the use of various barbiturate compounds has gained prominence and has practically displaced all other agents from the field of intravenous anesthesia. In 1929 sodium amytal was first used for intravenous anesthesia.^{8 9} Because of its prolonged action, it became less popular with the development of shorter acting barbiturates. Pentobarbital sodium (nembutal) was next introduced. It was found to be twice as potent as sodium amytal, with a much shorter recovery period. Considerable restlessness and the relatively long period of unconsciousness made both agents undesirable. Although extensively used in Europe, it was not until 1932 that evipal soluble (n-methyl-C-C-cyclo-hexamyl-methyl barbituric acid) was introduced to the medical profession of this country.^{10 11 12} When first introduced, this compound was unfortunately used as a single dose and fatalities were reported. The relaxation obtained was frequently poor.

Since 1934, pentothal sodium (sodium ethyl 1-methyl butyl thiobarbiturate) has been used extensively, and has largely replaced all other intravenous barbiturates. The chief advantages of pentothal sodium are that it is considerably more powerful than evipal sodium, giving

⁴ Lehrnbecher, A.: Experiences in 850 cases of intravenous anesthesia, *Arch. f. klin. chir.* **123**: 317-329, 1923.

⁵ Schnitzer, H.: Intravenous general anesthesia, *Munchen. med. Wchnschr.* **70**: 270-272, Mar. 1923.

⁶ Holtermann, C.: On intravenous avertin narcosis, *Schmerz Narkose-Anaesth.* **6**: 4, July 1933.

⁷ Frelenstein, W.: Intravenous avertin anesthesia according to Kirschner's method, *Klin. Wchnschr.* **9**: 742, Apr. 1930.

⁸ Hale, D. E.: Intravenous anesthesia, *Proc. Staff Meet. Mayo Clin.* **10**: 743, Nov. 1935.

⁹ Lundy, J. S.: Intravenous anesthesia: preliminary report of use of 2 new thiobarbiturates, *Proc. Staff Meet. Mayo Clin.* **10**: 536, Aug. 1935.

¹⁰ Livingston, E. M., Emy, S., and Lieber, H.: Evipal sodium: short intravenous anesthesia, *Am. J. Surg.* **26**: 516, Dec. 1934.

¹¹ Lundy, J. S.: Various anesthetic agents, especially some newer preparations, *Ill. M. J.* **70**: 134, Aug. 1936.

¹² Lundy, J. S. and Tovell, R. M.: Some of the newer local and general anesthetic agents, *Northwest Med.* **23**: 308, Sept. 1934.

fair relaxation, quick recovery and absence of restlessness in almost all cases.^{9 13}

During 1934, Nitzescu and Iacobovici published a report of their experiences with a solution of paraldehyde in isotonic dextrose by intravenous injection as a basal anesthetic.¹⁴ No untoward reactions were encountered in a small series of cases, but no further mention of this agent has been noted in recent literature, probably because of the simultaneous development of a number of very satisfactory barbitol compounds. Some consideration has been given in this country to the use of intravenous pentothal sodium as a basal anesthetic in conjunction with inhalation agents, although its usage in this manner has not been widely adopted.

No mention of the use of intravenous anesthesia in the statistics of surgical operations in the Annual Reports of the Surgeon General, U. S. Navy, is noted until 1935 when 5 short operations were reported to have been performed under intravenous evipal anesthesia. Subsequent annual reports for the calendar years of 1936 and 1937 list the use of evipal as an intravenous anesthetic, 6 and 18 times, respectively. The report for the year of 1938 lists the use of evipal in 4 instances and pentothal sodium in 2 instances. It is anticipated that the use of short acting barbiturates for intravenous anesthesia in the United States Navy will rapidly increase in popularity.

PHYSIOLOGICAL EFFECTS OF PENTOTHAL SODIUM

The action of pentothal sodium is directly on the higher cerebral centers as is the case with the mother substance, barbitol (di-ethyl barbituric acid). The drug is not excreted as such, but is apparently broken down in the liver and is rapidly eliminated from the body. The kidney function is not disturbed, and the blood chemistry is unchanged except for some increase in the blood sugar.¹⁵

The skin temperature of the extremities is normally increased from 1° to 2°F. The pulse varies from 80 to 120, depending on the depth of anesthesia. There is usually a transitory lowering of the blood pressure of from 10 to 20 millimeters of mercury, but prompt return to the preanesthetic level is the rule.

Muscular relaxation is general, but not sufficient to perform upper abdominal operations. Muscular twitching or slight convulsions occasionally occur, particularly if there is not a free airway. With deepening of anesthesia the respirations become more rapid and shallow. Conversely, with light anesthesia the respirations are slow and deep.

¹³ Lundy, J. S.: Intravenous anesthesia, *Am. J. Surg.* **34**: 559, Dec. 1936.

¹⁴ Nitzescu, I. I., and Iacobovici, J.: New method of general anesthesia with basal anesthesia by intravenous injection of glucosed paraldehyde, *Presse Med.* **42**: 331, Feb. 1934.

¹⁵ Lewis, A. E.: Intravenous pentothal sodium anesthesia, *Northwest Med.* **37**: 206, July 1938.

With light anesthesia, the patient may moan or cry out, but will not remember having felt pain due to temporary amnesia which is present for the period immediately preceding return to consciousness. Anesthesia is sufficiently deep for surgery when the supraorbital reflex is abolished. The pharyngeal reflex is not abolished during surgical anesthesia; consequently if liquids or foreign material enter the oropharynx, respirations ordinarily will stop and the patient will become cyanotic. Even with a good airway, a perceptible degree of cyanosis of the lips, fingertips, and mucous membranes may be evident.

DOSAGE

There is no set dosage for pentothal sodium. Age and weight are not important factors, although the aged and debilitated require smaller doses than usual.¹⁶ Chronic alcoholics and nervous or apprehensive patients require larger doses than the average patient. The nature of the operation and its duration naturally influence the amount used. The limits of dosage have been given as 0.1 gram for low to 4.0 grams for high, with periods of anesthesia varying from a few minutes to 4 hours or more. In general, 1.0 gram is considered the maximum dose for ordinary cases.¹⁷ Occasionally an individual will be encountered whose body will destroy the drug more rapidly than usual or the operation will take longer than expected. In these instances, following the use of 1.0 gram of pentothal sodium, supplementary inhalation anesthesia may be satisfactorily employed.

INDICATIONS

The indications for intravenous pentothal sodium anesthesia have not been uniformly classified and fully accepted by all surgeons. It has been used by various operators for virtually all types of operations, but it is best adapted for procedures which require only moderate muscular relaxation and a duration of not more than 30 minutes.

In view of the increasing frequency of recognized cases of degenerative encephalopathy following nitrous oxide anesthesia as presented by Courville¹⁸ and O'Brien and Steegmann,¹⁹ and following cyclopropane anesthesia as observed by Gebauer and Coleman,²⁰ and others, it seems likely that the use of intravenous anesthesia may further supplant inhalation anesthesia for operations of short duration.

¹⁶ Heard, K. M.: Pentothal: new intravenous anesthetic, *Canad. M. A. J.* 34: 628, June 1936.

¹⁷ Kassebohm, F. A. and Schreiber, M. J.: Intravenous anesthesia in obstetrics; comparative study of pentothal and evipal soluble with report of 250 cases, *Am. J. Surg.* 46: 577, May 1938.

¹⁸ Courville, C. B.: Pathogenesis of necrosis of cerebral gray matter following nitrous oxide anesthesia, *Ann. Surg.* 107: 371, Mar. 1938.

¹⁹ O'Brien, J. D. and Steegmann, A. T.: Severe degeneration of brain following nitrous oxide-oxygen anesthesia, *Ann. Surg.* 107: 486, Apr. 1938.

²⁰ Gebauer, P. W. and Coleman, F. P.: Post-anesthetic encephalopathy following cyclopropane, *Ann. Surg.* 107: 481, Apr. 1938.

Pentothal sodium causes no increase of mucus and does not irritate the respiratory system. It is indicated in acute respiratory tract infections wherein the irritating effects of inhalation anesthetic agents are not desired. For the incision and drainage of Ludwig's angina, intravenous pentothal sodium has recently received high praise.²¹ Tonsillectomy was performed under intravenous pentothal sodium anesthesia in a large selected series of children suffering with chronic respiratory infection, by Hutchinson and McHugh.²² These investigators were highly impressed by the safety and comfort of this method.

Because of safety from explosion, this form of anesthesia is well adapted to all types of cautery surgery.²³

Muscular relaxation is sufficiently good with pentothal sodium anesthesia for the performance of all types of perineal and anal surgery. It has been widely used in operative obstetrics with possibly the exception of version and Cesarean section.^{17 24} One distinct disadvantage in the use of this agent in uncomplicated labors is the tendency toward complete effacement of bearing down efforts. With ordinary dosage, no difficulty in establishing fetal respirations is encountered.

In cases of spinal anesthesia with novocain crystals where there is frequently a return of sensation before completion of operations which last longer than 1 hour, the intravenous injection of pentothal sodium has produced excellent supplemental anesthesia.

Intravenous pentothal sodium anesthesia is useful in controlling convulsions of eclampsia, meningitis and strychnin poisoning.²⁵ Convulsions occurring during inhalation ether anesthesia have been successfully treated with intravenous barbiturates and inhalations of high concentration oxygen.^{26 27}

When intra-urethral anesthesia is not satisfactory for cystoscopic examinations and manipulations, caudal, sacral and spinal anesthesia have frequently been employed. These agents were not entirely satisfactory, caudal anesthesia frequently producing priapism sufficient to interfere with instrumentation and distention of the bladder produced pain. Spinal anesthesia commonly causes an alarming drop in blood pressure, this being particularly undesirable in many elderly patients. Inhalation anesthesia has been undesirable because

²¹ Williams, A. C.: Ludwig's angina, *Surg. Gynec. & Obstet.* 70: 140-149, Feb. 1940.

²² Hutchinson, K., Mitchell, H. S. and McHugh, H.: Tonsillectomy under intravenous anesthesia in children suffering from chronic respiratory diseases, *Canad. M. A. J.* 30: 237, Sept. 1938.

²³ Organe, G. and Broad, R. J. B.: Pentothal with nitrous oxide and oxygen. *Lancet* 2: 1170-1172, Nov. 1938.

²⁴ Horsley, S.: Intravenous anesthesia for childbirth in a mental hospital, *Lancet* 230: 690, Mar. 1936.

²⁵ Tovell, R. M.: Modern trends in anesthesia, *California & West. Med.* 43: 192-196, Sept. 1935.

²⁶ Lundy, J. S.: Convulsions associated with general anesthesia, *Surgery* 1: 666-687, May 1937.

²⁷ Mousel, L. H.: Unusual case of convulsions under anesthesia, *Proc. Staff Meet. Mayo Clin.* 15: 33, Jan. 1940.

of fear and excitement during induction, and retching and vomiting during the postoperative period. For cystoscopic examinations, prostatic resections, removal of vesical tumors, urethral dilatation and all other transurethral manipulations which can be completed within 30 minutes, intravenous pentothal sodium is the anesthetic of choice.^{28 29} During the manipulation of a ureteral calculus by the transurethral route, the use of an anesthetic such as pentothal sodium given intravenously is wise practice according to Thompson, for ureteral spasm which will defeat one's effort will generally develop if an attempt is made without an anesthetic.³⁰

The closed treatment of many fractures, manipulation of joints, removal of metallic plates and nails, and other short orthopedic procedures are indications for this anesthetic.

In addition to its use for actual operations, it is very effective in relieving the patient during the dressing of large denuded surfaces, removal of packing, and other painful procedures.

In mental hospital practice, intravenous pentothal sodium anesthesia is being used to control manic excitement and to facilitate hypnosis as in narcoanalysis.³¹

CONTRAINDICATIONS

Intravenous pentothal sodium anesthesia is contraindicated in cases of jaundice or any marked hepatic insufficiency as indicated by the Quick hippuric acid test of liver function. Dehydration, marked hypotension, toxemia and sepsis are unfavorable for this anesthesia.^{32 32} This anesthetic is particularly contraindicated in all cardiorespiratory disorders in which dyspnea is a symptom, particularly in myocarditis and bronchial obstruction. It is not specially suited for most operations involving the upper respiratory passages, although it is adequate for certain operations on the larynx if preliminary cocaineization of the glottic region and vocal cords has been performed provided the proposed operation will result in a minimal amount of bleeding.

Due to the smallness of the respiratory passages, intravenous anesthesia is considered by some as not suitable for children. Opinion varies on the age limits.

Because of its sulfur content, the use of pentothal sodium has not been considered appropriate in cases which have received sulfanilamide or other sulfur containing compounds within 24 hours of the

²⁸ Thompson, G. J.: Transurethral surgery in 1935, *Proc. Staff Meet. Mayo Clin.* 11: 360, June 1936.

²⁹ Tovell, R. M. and Thompson, G. J.: Pentothal sodium anesthesia in urologic practice, *J. Urol.* 38: 81, July 1936.

³⁰ Thompson, G. J. and Kibler, J. M.: Treatment of ureteral calculus with particular reference to transurethral manipulation, *J. A. M. A.* 114: 6-12, Jan. 1940.

³¹ Horaley, S.: Pentothal sodium in mental hospital practice, *Brit. M. J.* 1: 938, May 1938.

³² Jarman, R. and Abel, A. L.: Intravenous anesthesia with pentothal sodium, *Lancet* 1: 422, Feb. 1936.

time of the operation. For such patients, the use of evipal sodium is the anesthetic of choice since it contains no sulfur.

Few people are sensitive to barbiturates, although the individual tolerance varies considerably. This anesthetic agent is obviously contraindicated in cases known to possess sensitivity to the drug.

PREPARATION OF THE PATIENT

Food and water should be withheld from the patient for a period sufficiently long to insure an empty stomach. Aspirated stomach contents, should vomiting occur, could easily produce serious respiratory embarrassment. Preferably the bladder and rectum should also be empty. When first introduced, premedication was omitted for operations to be performed under pentothal sodium anesthesia.^{9 11 32 33} There now appears to be general agreement on the desirability of always giving appropriate preliminary medication due to the decreased amount of the anesthetic required to produce anesthesia. Preoperative medication usually consists of pentobarbital sodium (nembutal) gr. $1\frac{1}{2}$ (0.097 gm.) by mouth, and morphine sulphate gr. $\frac{1}{4}$ (0.01) gm. with atropine sulphate gr. $\frac{1}{150}$ (0.0004 gm.) subcutaneously about 1 hour before the operation.

PREPARATION OF THE SOLUTION

Pentothal sodium is marketed in the powder form in ampules of 0.5 and 1.0 gram, respectively. It is readily soluble in water, and produces a clear, pale yellow, solution which has an odor of hydrogen bisulfide. The solution is relatively unstable, consequently a freshly prepared solution should be used for each patient. No special precautions need be followed in preparing the solution, except freshly distilled sterile water should be used and sterility of the mixing process should be maintained. The actual mixing may be completed in a medicine glass or beaker, or may take place in the ampule. Ordinarily 1.0 gram of the drug is dissolved in 20 cc. of water, thus producing a 5 percent solution. Recently it has been recommended that 40 cc. of water be used, making the concentration 2.5 percent, in order to prevent the appearance of an occasional delayed phlebitis at the site of venipuncture.³⁴ The solution is aspirated into an ordinary glass syringe of 20-cc. capacity to which a $1\frac{1}{4}$ -inch, 20-gauge needle with a short bevel is attached, and it is in readiness for injection.

³² Carraway, B. M.: Pentothal sodium with nasal oxygen: A report of 3,810 consecutive cases, *Anesth. & Analg.* 18:259, Sept.-Oct. 1939.

³⁴ Lundy, J. S., Tuohy, E. B., Adams, R. C., and Mousel, L. H.: Annual report for 1938 of section on anesthesia: including data on blood transfusion, *Proc. Staff Meet. Mayo Clin.* 14:273, May 1939.

ADMINISTRATION

Contrary to opinions expressed elsewhere, an expert anesthetist is not required for the administration of pentothal sodium. Any physician who will learn the physiological effects of the drug, select appropriate cases for its use, and will carefully and intelligently follow certain logical and widely accepted principles regarding the preparation of the patient and solution, dosage, and making the intravenous injection, should be able to satisfactorily administer this agent.

The usual site of venipuncture is the median basilic or median cephalic veins in the antecubital fossa, the dorsal metacarpal vein, or the great saphenous vein anterior to the medial malleolus. Varicose veins are not suitable for the venipuncture due to the uncertain venous circulation. While the solution is ordinarily injected into a vein, it may be introduced into the rubber tubing of a continuous venoclysis of physiological saline or other appropriate solution. Although the tissue reaction is not marked, special care must be exercised to prevent extravascular injection of the solution.

The injection is made intermittently rather than proceeding at a constant rate. The injection is not started until all is in readiness for the operation. The patient is asked to audibly count slowly as the injection is made. Two to three cc. of the 5-percent solution are injected in from 10 to 15 seconds, followed by a pause of about 30 seconds to allow the complete effect to appear. Relaxation appears before unconsciousness, and is evidenced by dropping of the jaw. If relaxation is not complete an additional 2 to 3 cc. of the solution may be injected followed by a pause of 30 seconds as before. The injection is continued intermittently until anesthesia is produced as manifested by relaxation, unconsciousness, and shallowness of respiration. There is no stage of excitement and the anesthesia resembles normal sleep.

If the jaw falls enough to interfere with respiration, a nurse should hold the jaw forward to insure a free airway. If there is more than very slight cyanosis, oxygen should be administered continuously with a face mask or intranasal catheter. Carraway²³ uses oxygen through an intranasal catheter, and has entirely eliminated symptoms of hiccough, respiratory depression, and cyanosis, according to his reports.

During the period of anesthesia the needle is allowed to remain in place in the vein, and 0.5 to 1 cc. of the solution is injected intermittently to maintain anesthesia at the desired level, the dose being regulated by the effect produced. In order to keep the needle free, minute quantities of solutions must be injected frequently.

Slight phonation, slight movement of the fingers or toes, and deepening respirations precede the return of consciousness.

To minimize the tendency to respiratory depression, 1 cc. of coramine (25 percent solution of pyridine betacarboxylic acid diethylamide) may be included in each 20 cc. of 2.5 percent pentothal sodium solution.^{11 35} The increased depth and rate of respiration appears to be analagous to the use of carbon dioxide during inhalation anesthesia. This technic is used routinely by some anesthetists, particularly where strong preliminary medication has been used.

To facilitate the observation of the depth and rate of respiration, Lundy devised the use of a small wisp of cotton in the shape of a butterfly, which is attached to the upper lip with a small piece of adhesive tape.³⁵ One wing of the "butterfly" overhangs the nostrils and lips respectively, and respiratory movement of air will cause the fibers of the cotton to be set into motion. If oxygen is being administered simultaneously, the butterfly is discarded, and respiratory movements are observed in the bag of the gas machine.

Intravenous pentothal sodium anesthesia may be used in the office, but should not be used unless oxygen and carbon dioxide mixtures are available for inhalation and possible artificial respiration.³⁶ When administered to ambulant patients, they should be required to remain under observation until ataxia has disappeared and then should not be allowed to drive a car or go home alone.

In addition to oxygen and carbon dioxide mixture for inhalation, certain stimulants as coramine, metrazol, picrotoxin, or adrenalin should be available for intravenous or intracardiac injection for serious respiratory depression.^{32 37}

Recovery from pentothal sodium anesthesia is gradual and is associated with a variable degree of amnesia. The recovery period varies from a few minutes to as much as an hour, depending on the length and degree of anesthesia. Morphine sulphate should not be administered following the anesthetic until the patient is fully conscious. If sedation is imperative for restlessness or apparent actual pain, fractional doses of morphine, $\frac{1}{4}$ grain, should be given and repeated 1 hour later if necessary. This measure will serve to reduce the excessive depression which has been seen and which has been a factor in unfavorable criticism of this form of anesthesia. Special attention should be given to elderly patients and those in whom anesthesia has been maintained for 1 hour or more.

¹¹ Lundy, J. S.: Method of minimizing respiratory depression when using soluble barbiturates intravenously, *Proc. Staff Meet., Mayo Clin.* 10: 791, Dec. 1935.

³⁵ Pratt, T. W., Tatum, A. L., Hathaway, H. R. and Waters, R. M.: Sodium ethyl (1-methyl butyl) thio-barbiturates; preliminary experimental and clinical study, *Am. J. Surg.* 31: 464, Mar. 1936.

³⁷ Carraway, B. M. and Carraway, C. N.: Intravenous anesthesia; clinical study of 1,900 cases, *Am. J. Surg.* 30: 576, Mar. 1938.

ILLUSTRATIVE CASES

Case 1.—C. B. F., male, age 38, file No. 23143. Admitted, July 22, 1939.

DIAGNOSIS: Cholecystitis, chronic; cholelithiasis; appendicitis, chronic.

OPERATION: On September 13, 1939, appendectomy and cholecystectomy were performed. The operation was started under spinal anesthesia of novocain crystals 200 mg., but sensation returned after about 1 hour. Intravenous pentothal sodium, 1.0 gm., produced satisfactory supplemental anesthesia for a period of 50 minutes. Operation was still uncompleted, inhalation ether anesthesia being employed for the remaining steps of the operation.

RESULTS: No postoperative complications. Discharged from hospital well on October 22, 1939.

Case 2.—P. R. Le V., male, age 54, file No. 23210. Admitted, September 27, 1939.

DIAGNOSIS: Hernia recurrent after operation, left inguinal direct.

OPERATION: On September 29, 1939, hernia repair was performed with a modified Bassini technic and implantation of a strip of fascia lata. The operation was started under spinal anesthesia of novocain crystals, 200 mg., but patient complained of pain after 1 hour and 10 minutes. The operation was completed with the intravenous injection of pentothal sodium, 0.6 gm., which produced supplemental anesthesia for a period of 45 minutes.

RESULTS: No postoperative complications. Transferred to sick leave on January 9, 1940, and has subsequently returned to his regular duties.

Case 3.—J. W., female, age 22, file No. 23214. Admitted, September 27, 1939.

DIAGNOSIS: Displacement, uterus (retroversion); fibrocystic left ovary; par-ovarian cyst, left; salpingitis, chronic, left; appendicitis, subacute, mild.

OPERATION: On September 28, 1939, appendectomy, left salpingo-oophorectomy, and a Gilliam uterine suspension were performed. The operation was started under spinal anesthesia of novocain crystals, 150 mg., but sensation returned after 1 hour and 25 minutes. The operation was completed under intravenous injection of pentothal sodium, 1.0 gm., which produced supplemental anesthesia for 20 minutes.

RESULTS: No postoperative complications. Discharged from hospital October 22, 1939, to home for further convalescence.

Case 4.—J. W. N., male, age 43, file No. 23223. Admitted, October 8, 1939.

DIAGNOSIS: Ulcer, duodenum, with obstruction.

OPERATION: On November 7, 1939, a partial gastrectomy with posterior Polya anastomosis was performed. The operation was started under spinal anesthesia of novocain crystals, 200 mg., but sensation returned after 1 hour and 35 minutes. Intravenous pentothal sodium anesthesia, 0.4 gm., produced anesthesia for a period of about 10 minutes. Because of unsatisfactory muscular relaxation, the remainder of the operation was performed under inhalation ether anesthesia.

RESULTS: No postoperative complications due to anesthesia. Returned to duty, much improved, on January 10, 1940.

Case 5.—L. R., female, age 28, file No. 23254. Admitted, October 25, 1939.

DIAGNOSIS: Displacement, uterus (retroversion and retrocession); old laceration of cervix with papillary erosion and fibrosis; old, incompletely repaired, third-degree laceration of perineum.

OPERATION: On October 26, 1939, complete hysterectomy was performed followed immediately by perineal repair. The hysterectomy was started under spinal anesthesia of novocain crystals, 200 mg., but sensation returned after 1 hour and 10 minutes. The hysterectomy was completed under supplemental

intravenous pentothal sodium, 0.8 gm., lasting 50 minutes. The second operation, perineorrhaphy, was then performed under inhalation ether anesthesia.

RESULTS: No postoperative complications. Discharged to home in good condition on November 15, 1939, for further convalescence.

Case 6.—E. F., female, age 31, file No. 23260. Admitted, October 29, 1939.

DIAGNOSIS: Cholecystitis, chronic; cholelithiasis.

OPERATION: On October 31, 1939, cholecystectomy was performed. The operation was commenced under spinal anesthesia of novocain crystals, 200 gm., but sensation returned after about 1 hour and 10 minutes. Supplemental anesthesia for 10 more minutes was obtained by the intravenous injection of pentothal sodium, 0.25 gm., but was discontinued because of the inability to maintain a continuous intravenous injection due to poor veins. The operation was completed under ether anesthesia.

RESULTS: No postoperative complications. Discharged to home on November 16, 1939, for further convalescence.

Case 7.—A. M. R., male, age 17, file No. 23311. Admitted, November 30, 1939.

DIAGNOSIS: Palmar abscess, left hand.

OPERATION: On December 3, 1939, the palmar abscess was incised and drained under an intravenous pentothal sodium anesthetic, 0.25 gm. No preliminary medication was used.

RESULTS: Returned to duty, well, on December 22, 1939.

Case 8.—I. R., female, age 37, file No. 23312. Admitted, December 1, 1939.

DIAGNOSIS: Pregnancy at term; marginal placenta praevia with recent hemorrhage.

OPERATION: Classical Cesarean section was performed on the day of admission under field block anesthesia. During closure of parietal peritoneum, patient suffered pain. This was relieved by the intravenous injection of pentothal sodium, 0.1 gm., and the remainder of the abdominal wall was closed under the original field block anesthesia.

RESULTS: No postoperative complications. Mother and infant discharged to home in good condition, December 11, 1939.

Case 9.—G. M. D., female, age 54, file No. 23329. Admitted, December 7, 1939.

DIAGNOSIS: Calculus, left ureter.

TRANSURETHRAL MANIPULATIONS: Cystoscopic examination and kidney study performed without anesthesia on December 12, 1939, revealed a calculus impacted in the terminal portion of the left ureter. On December 14, 1939, without preliminary medication, transurethral manipulation was performed under intravenous pentothal sodium anesthesia, 0.7 gm., which lasted 27 minutes. On December 28, 1939, and January 8, 1940, transurethral manipulations were again performed under intravenous pentothal sodium anesthesia. Preliminary medication consisting of the hypodermic injection of morphine sulphate, gr. 1/6, and atropine sulphate, gr. 2/300, 1 hour before each treatment. Anesthesia was obtained for 29 minutes by 0.45 gm., and for 40 minutes by 0.75 gm., of the drug respectively.

On January 18, 1940, a final manipulation of the ureteral calculus was performed. Premedication 1 hour before the treatment consisted of pentobarbital sodium (nembutal), gr. 1½, by mouth, and the hypodermic injection of morphine sulphate, gr. ¼, and atropine sulphate, gr. 1/150. The intravenous injection of pentothal sodium, 1.0 gm., produced anesthesia for 1 hour and 9 minutes.

RESULTS: No complications during or following any manipulation. X-Ray examination revealed that the calculus had passed and patient was returned to duty on January 26, 1940.

Case 10.—D. S., female, age 44, file No. 23480. Admitted, February 11, 1940.
DIAGNOSIS: Carcinoma, cervix.

OPERATION: On February 13, 1940, complete hysterectomy was performed under spinal anesthesia, 200 gm. novocain crystals, but sensation returned after 1 hour and 25 minutes. The operation was completed with the intravenous injection of pentothal sodium, 1.0 gm., which produced supplemental anesthesia for a period of 54 minutes.

RESULTS: No postoperative complications. Patient still in hospital. Operation performed after original presentation of paper.

Case 11.—T. P. F., male, age 21, file No. 23493. Admitted, February 17, 1940.
DIAGNOSIS: Appendicitis, chronic, retrocecal.

OPERATION: On February 17, 1940, appendectomy was performed. The operation was started under spinal anesthesia of novocain crystals, 150 mg., sensation returning after 50 minutes. The operation was completed with the intravenous injection of pentothal sodium, 1.3 gm., which produced supplemental anesthesia for a period of 35 minutes.

About 10 days postoperatively there was some elevation of temperature and moderate purulent drainage appeared from the operative wound. Because of evidence of a retroperitoneal abscess, exploratory operation was performed on March 1, 1940. This operation was started under spinal anesthesia of novocain crystals, 150 mg., but sensation returned after about 35 minutes. The operation was completed in an additional period of supplemental anesthesia lasting 26 minutes, using 1.0 grams of pentothal sodium intravenously.

RESULTS: Patient is convalescing satisfactorily. Operations performed after original presentation of paper.

Case 12.—C. C. R., male, age 53. Outpatient.

DIAGNOSIS: Moderate arterial hypertension; nonvital teeth. History of previous reaction to procaine-epinephrin mixture during dental treatment.

OPERATIONS: Dental extractions were performed on January 12, 1940, January 24, 1940, and January 31, 1940, using 0.25, 0.5, and 0.4 gm., of pentothal sodium for intravenous anesthesia. Preliminary medication was omitted for the first extraction, but was used for the second and third procedures.

RESULTS: No material change in blood pressure. Not hospitalized.

Case 13.—C. M. S., young adult male. Outpatient.

DIAGNOSIS: Alveolar abscess.

OPERATION: Conduction anesthesia unsuccessful and local infiltration not considered desirable. Dental extraction performed on December 5, 1939, under intravenous injection of 0.3 gm. pentothal sodium. No preliminary medication.

RESULTS: No complications. Patient not hospitalized.

Case 14.—N. H., female, age 21. Outpatient.

DIAGNOSIS: Papillary erosion and fibrosis of cervix.

OPERATION: Painless biopsy performed under anesthesia produced by the intravenous injection of 0.3 gm., pentothal sodium. Premedication used.

RESULTS: No complications. Not hospitalized.

Case 15.—L. S., female, age 34, file No. 23529½. Admitted, March 4, 1940.

DIAGNOSIS: Endometritis.

OPERATION: On March 5, 1940, dilatation of the cervix and curettage of the uterus was performed under intravenous pentothal sodium anesthesia, using 0.9 grams over a period of 15 minutes.

RESULTS: No complications. Patient operated upon subsequent to presentation of original paper.

CASE 16.—A. H., female, age 30, file No. 23530. Admitted, March 5, 1940.

DIAGNOSIS: Cyst, vagina.

OPERATION: Resection of vaginal cyst performed on March 6, 1940. The operation was started under spinal anesthesia of novocain crystals, 150 mg., but sensation returned after 55 minutes. The operation was completed in an additional period of 35 minutes during which time supplementary anesthesia was produced by the intravenous injection of 0.37 grams of pentothal sodium.

RESULTS: No complications. Patient operated upon subsequent to presentation of original paper.

SUMMARY OF ANESTHETIC AGENTS USED IN ILLUSTRATIVE CASES

Spinal novocain plus intravenous pentothal sodium

Repair of recurrent hernia.....	1
Appendectomy, left salpingo-oophorectomy, and uterine suspension.....	1
Complete hysterectomy.....	1
Appendectomy.....	1
Drainage of retroperitoneal abscess.....	1
Resection of vaginal cyst.....	1

Spinal novocain, intravenous pentothal sodium and inhalant ether

Cholecystectomy.....	2
Partial gastrectomy, Polya type.....	1
Complete hysterectomy.....	1

Field block with procain 1 percent and intravenous pentothal sodium

Caesarean section.....	1
------------------------	---

Intravenous pentothal sodium anesthesia alone

Incision and drainage of palmar abscess.....	1
Transurethral manipulation.....	4
Dental extractions.....	4
Biopsy of cervix.....	1
Dilatation and curettage.....	1

Total.....	22
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COMMENTS ON ILLUSTRATIVE CASES

Even with a dose of 200 mg. of novocain crystals, spinal anesthesia cannot be expected to last much longer than 1 hour, unless more toxic spinal anesthesia agents are used. Cases 1, 2, 3, 4, 5, 6, 10, 11 (2 operations), and 16, represent major surgical procedures which were started under novocain spinal anesthesia, but supplementary anesthesia was required due to early return of sensation. In cases 2, 3, 5, 10, and 11, supplemental intravenous pentothal sodium was sufficient, but in cases 1, 4, and 6, additional inhalant ether anesthesia was required. In case 4, a partial gastrectomy, intravenous anesthesia produced insufficient muscular relaxation, while in case 6, intravenous

anesthesia was abandoned early due to inability to maintain continuous intravenous injection because of poor veins. In case 8, intravenous pentothal sodium was used to supplement an abdominal field block for Caesarean section, the injection having been started following delivery of the infant.

In general, pentothal sodium was considered to be entirely satisfactory as a supplemental anesthetic following spinal anesthesia, although in certain cases subsequent inhalation anesthesia was required. Intravenous pentothal sodium anesthesia was most satisfactory for transurethral manipulation, dental extraction, biopsy, and other short procedures. It was most satisfactory when preceded by appropriate preliminary medication. It is to be emphasized that pentothal sodium was used in each instance by the intermittent or fractional method of continuous injection as previously described.

COMPLICATIONS

With any anesthetic agent, overdose results in a complication. An overdose of pentothal sodium results in a central respiratory paralysis with subsequent circulatory failure. The heart may continue to beat for 2 to 3 minutes following cessation of respiration.¹⁵

Occasionally jerky clonic muscular tremors of the extremities occur, particularly when the airway is not free. Rarely is there a postoperative headache. Occasionally there is a state of restlessness during recovery.

The use of intravenous pentothal sodium anesthesia does not seem to be associated with an increased frequency of postoperative pulmonary complications such as atelectasis and pneumonia, despite varying degrees of respiratory depression and shallow breathing.

MORTALITY

Positive statistical data on the mortality rates of intravenous pentothal sodium anesthesia are lacking because of the relative recent development of this agent. Published reports on this subject are, however, uniformly enthusiastic in their praise for pentothal sodium as a short-acting anesthetic in selected cases. An analysis of the fatalities reported at the time of the introduction of intravenous barbiturates to the field of anesthesia invariably shows the use of a single injected dose of the drug as opposed to the present-day accepted method of intermittent fractional injection. Injudicious use of morphine before complete recovery from barbiturate anesthesia, thereby causing profound respiratory depression, doubtlessly contributed to some deaths. With our present knowledge of the action and limitations of pentothal sodium, mortality due to the drug itself should be practically nil. Table 1 represents a summary of the

published observations of five different observers totalling 9,285 intravenous pentothal sodium anesthetics with 6 deaths, a mortality rate of 0.064 percent.

TABLE 1.—*Mortality of intravenous pentothal sodium anesthesia*

Reference	Number of cases	Deaths	Mortality per cent
Lewis: ¹⁶			
Cameron's series.....	225	0	0
Tovell's series.....	4,000	6	0.15
Kassebohm and Schreiber ¹⁷	250	0	0
Jarman ¹⁸	1,000	0	0
Carraway and Carraway ¹⁹	3,810	0	0
Total.....	9,285	6	.064

SUMMARY

Pentothal sodium has been found to be a highly satisfactory agent for many cases where a general anesthetic is required. It is best suited for adult patients when the duration of the examination or operative procedure is expected to be 30 minutes or less. It is very valuable when a supplementary anesthetic is needed at the end of a local or spinal anesthetic.

With intelligent selection of case material, a thorough knowledge of the physiological action of the drug, and when given by the current method of intermittent injection, any physician should be able to use pentothal sodium as an intravenous anesthetic without difficulty. The maximum safe dose is considered to be 1.0 gram.

THE BASIS OF SUCCESSFUL PARENTERAL THERAPY ¹

By Carl W. Walter, M. D.

The parenteral administration of medicine has become so commonplace that clinicians are but rarely involved in the preparation of solutions and apparatus used for such therapy. Accordingly, the introduction of a new drug, an unusual case, or an untoward reaction often confronts the clinician with technical problems which he does not understand. Those who prescribe parenteral therapy should be familiar with the basic principles which determine its success. There is no excuse for untoward reactions due to routine therapy. A new drug or an unusual case may demand an improvised technic for the preparation of suitable solutions, but the same principles which protect the ordinary patient must provide the bases for the new technic if it is to be safe and successful.

¹ From the Laboratory for Surgical Research, Harvard Medical School and the Surgical Clinic of the Peter Bent Brigham Hospital, Boston, Mass.

Untoward reactions following the injection of sterile "distilled" water are characterized by symptoms suggestive of protein shock. The mild forms are evidenced merely by a moderate elevation of temperature which reaches a peak and returns to normal within 6 hours following the infusion. In more severe reactions hyperpyrexia follows chills or pain in the back or legs and usually occurs while the infusion is still flowing. Nausea, vomiting, and diarrhea may ensue, perhaps associated with a fall in blood pressure. In serious reactions a marked fall in blood pressure is associated with cyanosis or circulatory collapse which may result in death. Mild reactions are distressing to the patients; severe reactions are dangerous, particularly since the indications for this form of therapy may present a patient in critical condition.

The cause of such reactions has been the subject of much conjecture despite clarifying laboratory and clinical evidence. Wechselmann,² 1911, noticed that distilled water acquired, on standing, the property of producing febrile reactions. He believed bacterial contamination responsible and advocated the administration of freshly distilled water only. Müller,³ 1911, described the bacterial flora of numerous samples of distilled water. Hort and Penfold,⁴ 1911, confirmed Wechselmann's observation that freshly distilled water became contaminated with bacteria. Removal of these bacteria by Birkefeld filtration did not eliminate the reactions. Thirteen types of bacteria were identified as capable of imparting the pyrogenic property to distilled water.

Siebert^{5, 6, 7} and her collaborators, 1923-27, added other organisms to the list and were able to differentiate three groups of pyrogenic bacteria: those provoking low fever, those causing chills and high fever, and those responsible for immediate prostration and death. The etiologic factor was a filterable, thermostable, desiccation resistant exotoxin produced by various organisms capable of growing in distilled water. The contamination of distilled water was due to air-borne bacteria and/or a pyrogenic exotoxin carried from the raw water in the generator of the still to the condensers by entrainment. Distillation in a still designed to prevent entrainment, immediate sterilization after distillation, and sealing to protect sterility were recommended as essential steps in providing safe water for parenteral injection.

Another source of pollution of solutions was shown to be pyrogenic exotoxin which dried on the inner surface of glassware. Flasks in

² Wechselmann: Neuere Erfahrungen über Intravenöse Salvarsaninjektionen ohne Reaktionserscheinungen. München. Med. Wchnschr. 88: 1510, 1911.

³ Müller, P. T.: Ueber den Bakteriengehalt des in Apotheken erhältlichen destillierten Wassers. München. Med. Wchnschr., 88: 2739, 1911.

⁴ Hort, E. C., and Penfold, W. J.: Dangers of saline injections, Brit. Med. J. 2: 1589, 1911.

⁵ Siebert, F. B.: Fever-producing substance found in some distilled waters. Am. J. Physiol., 67: 90, December 1923.

⁶ Siebert, F. B.: Cause of many febrile reactions following intravenous injections. Am. J. Physiol., 71: 621, February 1925.

⁷ Siebert, F. B., and Mendel, L. B.: Protein fevers; with special reference to casein. Am. J. Physiol., 67: 105, December 1923.

which pyrogenic water had evaporated could be rendered safe by merely rinsing them with freshly distilled water.

Banks⁸ showed that pyrogen could be destroyed by heating at 284° F. (38 p.s.i. gauge) for 30 minutes. Co Tui⁹ and his co-workers found that the pyrogen could be absorbed on Seitz asbestos filters and estimated the size of the pyrogenic particle to be between 50 millimicrons and 1 micron. These facts explain many difficulties encountered in parenteral administration, not only of electrolytes and dextrose, but also of arsenicals, infiltration anesthetics, contrast media, and citrated blood or plasma.

There are but two requisites for a safe supply of parenteral solutions—a source of pure raw materials and centralized responsibility for cleanness in the preparation of solutions and apparatus. Any hospital where major surgery is performed has the necessary sterilizing equipment and the trained personnel to whom such responsibility can be safely delegated.

DISTILLED WATER

The quality of freshly distilled water is affected by two factors—the design of the still and the care and intelligence with which it is maintained and operated. Because those entrusted with the maintenance and/or operation of stills often have little concept of how distillation is accomplished or what precautions must be taken to obtain pure distillate, various protective features have been incorporated in the design of stills. Singly distilled water is sufficiently pure for intravenous work unless the water supply is unusually bad or the personnel is unreliable or shifted so often that no one is familiar with the still. Double distillation provides a factor of safety in such instances because faulty adjustment of the steam or water supply, failure to clean the generator, or operation beyond the normal capacity of the still are not so likely to result in pollution of the final distillate. However, a well-designed still, intelligently operated and carefully maintained, will deliver in one distillation, water as pure as that obtained by double or triple distillation.

Diagrammatic elevations of the various types of still in hospital use are illustrated in figures 17, 18, and 19. Although the design varies, certain common elements can be noted. The source of heat, a steam coil is illustrated, should be accurately controlled to insure constant heat input. Evaporator design should provide adequate space for the slow, vertical rise of steam to permit droplets to fall out.

Condensers should be lined with metal which resists attack by distilled water. Pure tin is widely used. The condensers of a well

⁸ Banks, H. M.: Study of hyperpyrexia reaction following intravenous therapy. *Am. J. Clin. Path.*, 4: 260, May 1934.

⁹ CoTui, F. W., McCloskey, K. L., Schrift, M., and Yates, A. L.: New method of preparing infusion fluids. *J. A. M. A.* 100: 250, July 1937.

functioning still need never be cleansed. Indeed, cleansing is likely to introduce dirt or scratch the protective surfaces.

The chief cause of pollution of the distillate is entrainment of droplets of spray or foam thrown off by the boiling water. Thus, particles (bacteria or spores) and/or solutes (pyrogens) are carried from the raw water in the generator to the condenser by the steam. Stills should be designed to eliminate entrainment. (Fig. 17, 18, 19.)

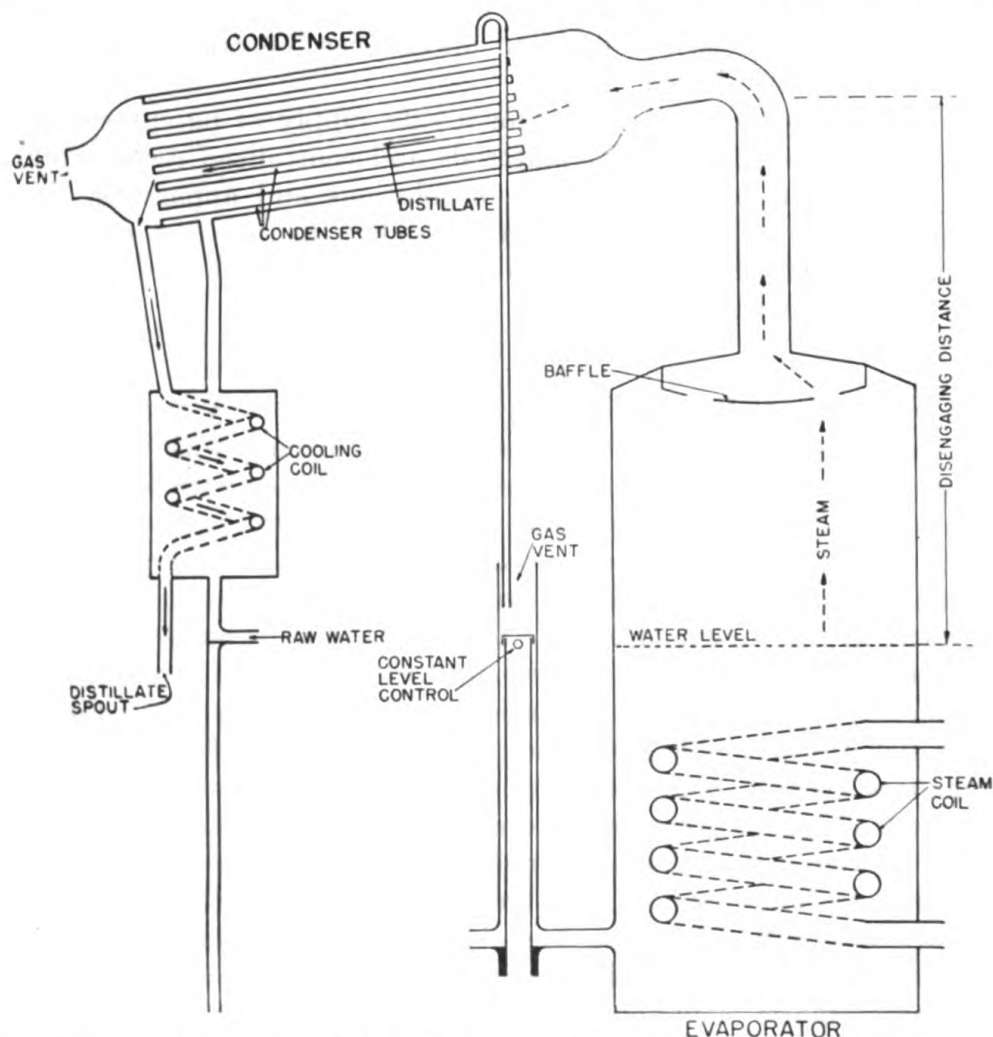


FIGURE. 17.—Diagrammatic elevation of a still illustrating baffles and condenser.

Properly placed baffles contribute much to the purity of the distillate by preventing pollution of the condensers with spray splashed from the surface of the boiling water. If well designed, baffles eliminate entrainment by causing particles to impinge thus removing them from the steam. The scrubbing action obtained by condensing a portion of the steam in a primary condenser is also helpful. Location of the condensers at a distance, preferably vertically, from the boiling surface permits many droplets to fall out of the steam. Partial chilling of the steam causes condensation of small droplets, making

them heavy enough to fall back. The passage of the steam through multiple tortuous baffles upon which the droplets impinge and are removed is a positive guard against entrainment. Entrainment due to the foaming of hard water can be avoided by preventing the concentration of impurities in the generator. A deconcentrator or bleeder will permit continuous operation of a generator without harmful concentration of residual impurities.

Gross priming of a still can be prevented by careful control of flow of water and the rate of heating. Water supply must be controlled to insure uniform flow of water through the evaporator, otherwise priming occurs whenever the water pressure drops. Too rapid heating causes explosive ebullition of steam and much splashing which can be eliminated to a large degree by providing a large surface for

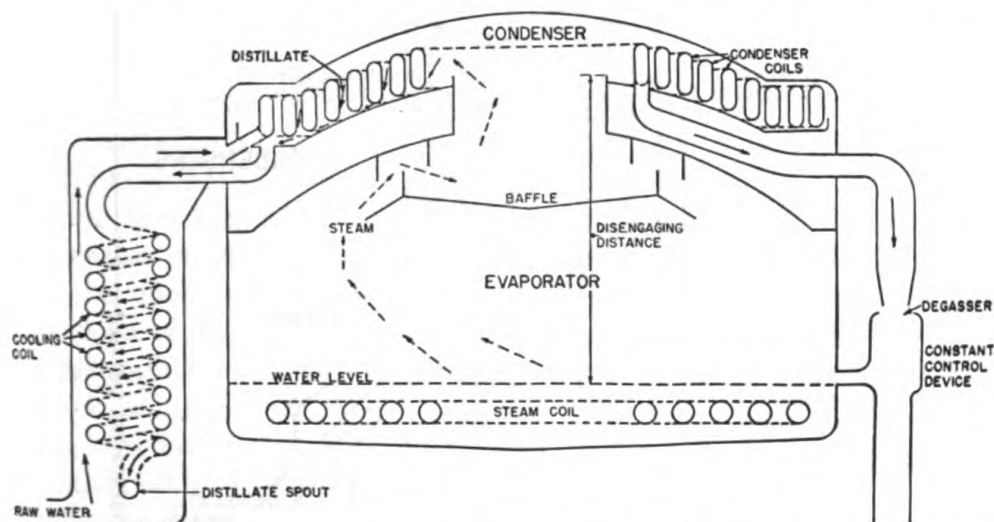


FIGURE. 18.—Diagrammatic elevation of a still illustrating baffles and condenser.

evaporation and avoiding deep water above the heat exchanging surfaces. The increased velocity of the stream of steam carries the splashed droplets past baffles or traps which are ordinarily effective. Because other users of water or steam frequently cause great fluctuations in the pressure and hence in the flow of these supplies, automatic control must often be relied upon to prevent priming. Overloaded electrical circuits may also influence the quality of the distillate if the still should be adjusted while the voltage is low.

Chemically pure distilled water cannot be stored unless it is hermetically sealed in sterile containers; hence, distilled water must be collected in a storage tank just large enough to contain a working supply. An inverted pyrex carboy fitted with a glass stopcock is best, because such a container can be drained dry so that bacteria cannot grow in residual water and pollute subsequent collections of distillate. Such carboys should be drained as soon as there is no immediate use for distilled water.

The purity of water can be measured readily by determining its conductivity. The distillate from a well designed, properly operated still should have a maximum conductivity of 2×10^{-6} mhos at 20°C . One part per million of chloride ion will increase the conductivity of

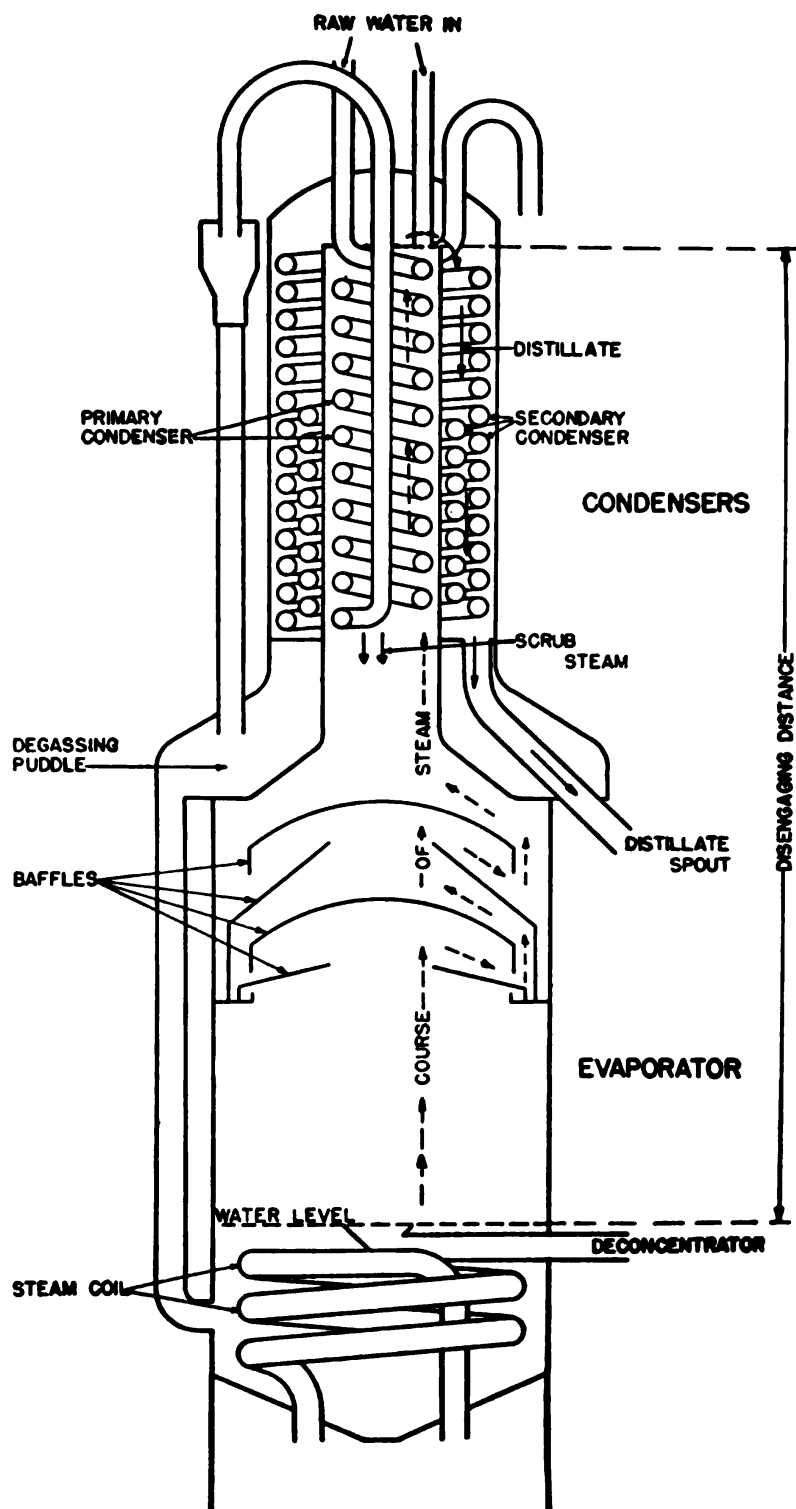


FIGURE 19.—Diagrammatic elevation of a still illustrating baffles and condenser.

distilled water about 50 percent. The presence of electrolytes in freshly distilled water indicates contamination with tap water by entrainment or leakage from a faulty condenser. It may then be assumed that the water has also been contaminated by pyrogenic substances accompanying the electrolytes. Some stills produce impure water sporadically and should be equipped to check the purity of the distillate continuously.

Biological methods must be employed to identify actual pyrogen content. The simplest test is that of injecting 10 cc. of the questionable solution into the ear vein of a rabbit and determining the rectal temperature hourly for 3 or 4 hours. The rabbit's normal temperature ranges from 101.2° to 103° F. under standardized conditions. The febrile reaction which results from the injection of pyrogen raises this to 105–107°. ^{8 10}

GLASSWARE

Pyrex glassware is the most satisfactory not only because of its high resistance to mechanical and thermal shock, but also because its stable annealed surface resists hydrolysis by the solutions. The alkaline film formed at the glass-liquid interface of a soft glass flask may cause polymerization of the dextrose during sterilization.

To be chemically clean, glassware must be freed of the initial soil as well as of insoluble deposits (fig. 20) resulting from the interaction of washing agents (soaps and detergents) and water or soil containing more than traces of alkaline earths (Ca and Mg).¹¹ The white opalescent film which becomes noticeable as glassware dries is due to insoluble alkali earth soaps formed by this interaction. Dried blood (fig. 21) and closely adherent bacterial growths (fig. 22) are the usual types of soil encountered. Blood and residual solutions invite and support bacterial life and may be the cause of pollution with pyrogen. Dried fungi are particularly difficult to remove. They may be invisible until hydrolized during sterilization, at which time they swell and become opaque, and may be mistaken for wisps of cotton floating in the solution. Because glassware is often used in the laboratory or utility room, residues of feces, urine, pus, and transudates are often encountered. The most tenacious soil is a greasy film which accumulates when the glassware is used repeatedly without adequate cleansing.

After glassware has been thoroughly cleansed,¹² it must be rinsed with freshly distilled water to remove any pyrogen contaminating

⁸ Seibert, F. B., and Mendel, L. B.: Temperature variations in rabbits. *Am. J. Physiol.*, 67: 83, December 1923.

¹¹ Hall, G. O., and Schwartz, C.: Sanitary value of sodium metaphosphate in dishwashing. *Ind. & Eng. Chem.*, 30: 421, April 1937.

¹² Walter, C. W.: Relation of proper preparation of solutions for intravenous therapy to febrile reactions. *Ann. Surg.*, 113: 4, 603., October 1940.

its inner surface and positioned to drain as well as to protect the inside from dust. When distilled water is poured from a clean flask, a film of water is left spread on the inner surface. This film breaks immediately and forms droplets wherever greasy soil alters the surface. Clean glass should show no "water breaks" (fig. 23) in the film of distilled water left after the final rinsing, and it should be crystal clear when dry.

RUBBER TUBING

In selecting rubber tubing for use in parenteral therapy, it is essential that the inner surface be free from pits, wrinkles, and mold marks (fig. 24), where blood and bacterial residues may lodge and make cleaning difficult. The surface of rubber exposed to the fluid can be decreased markedly by selecting a tubing of small inside diameter ($\frac{1}{8}$ inch). A small lumen also facilitates the expulsion of air from the system, since fluid runs through it as a solid column rather than trickling down one side. Any nontoxic rubber can be used, but a rubber compounded to insure maximum heat resistance is more economical. Commercially available tubing will withstand 75 sterilizations before losing its elasticity.

INTRAVENOUS KITS

PREPARATION.—The establishment of a source of chemically pure parenteral fluids is futile unless an equally safe supply of apparatus for its administration is constantly available. Bacterial growth in residual solution or blood in such equipment produces pyrogen which is leached out of the dried deposits on the inner wall during the administration. This source of pyrogen can be eliminated only by conscientious cleaning of the inner surfaces of tubing, needles, and adapters. This cleansing must be done immediately before sterilization, because bacteria are likely to grow in the moisture left in the apparatus to insure sterilization.

The rubber tubing is cleaned by flushing the lumen with hot detergent solution. (Any dishwashing compound which leaves glassware crystal clear on drying can be used. Solutions containing soap should be avoided.) Freshly distilled water is then run through the tubing for 10 seconds to remove the detergent and to leave the inner surface chemically clean. Excess water is drained off. No further attempt is made to dry the lumen of the tubing.

Needles are cleaned by reaming them with a snugly fitting stilet and forcing hot detergent solution through the cannula. They are rinsed with distilled water and inspected critically, not only for cleanness and sharpness, but also for weakness which might cause unexpected breakage. This is done by holding the hub firmly while the tip is sprung through an arc. The weakened ones will snap off or bend at the junction of the hub and needle. Needles should not

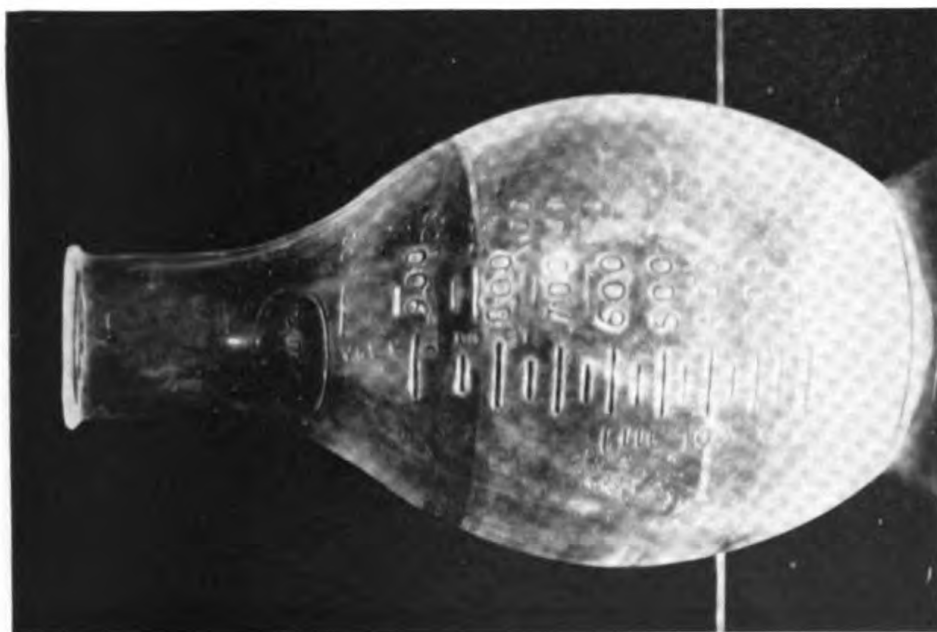


FIGURE 20.—ILLUSTRATION SHOWING INSOLUBLE DEPOSITS IN FLASK.

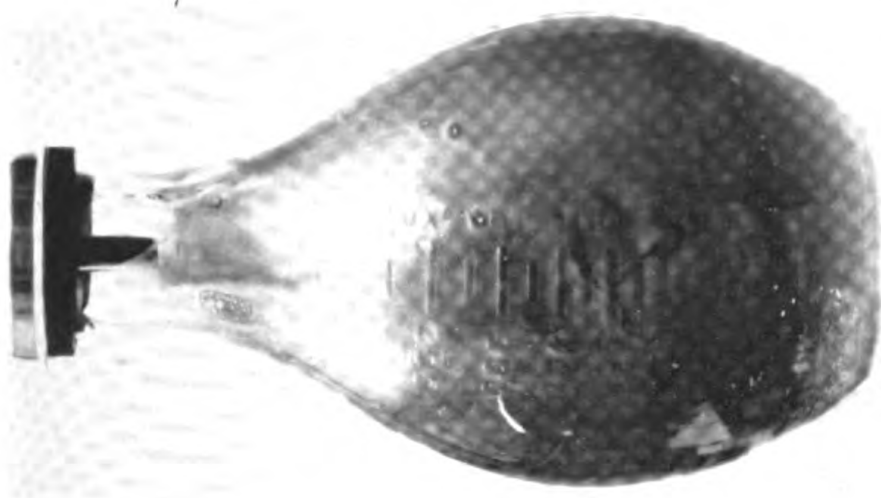


FIGURE 21.—FLASK SHOWING DRIED BLOOD.

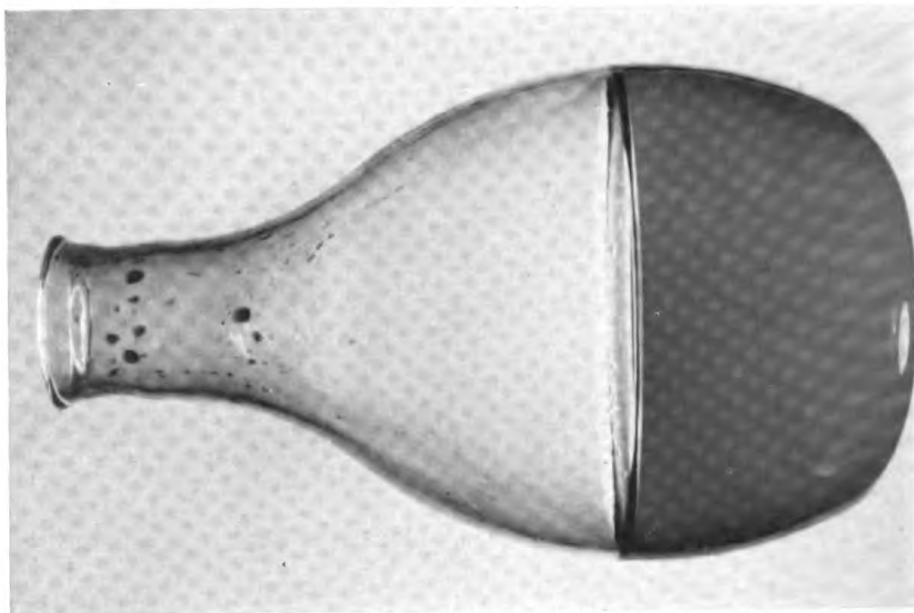


FIGURE 23.—FLASK SHOWING "WATER BREAKS."

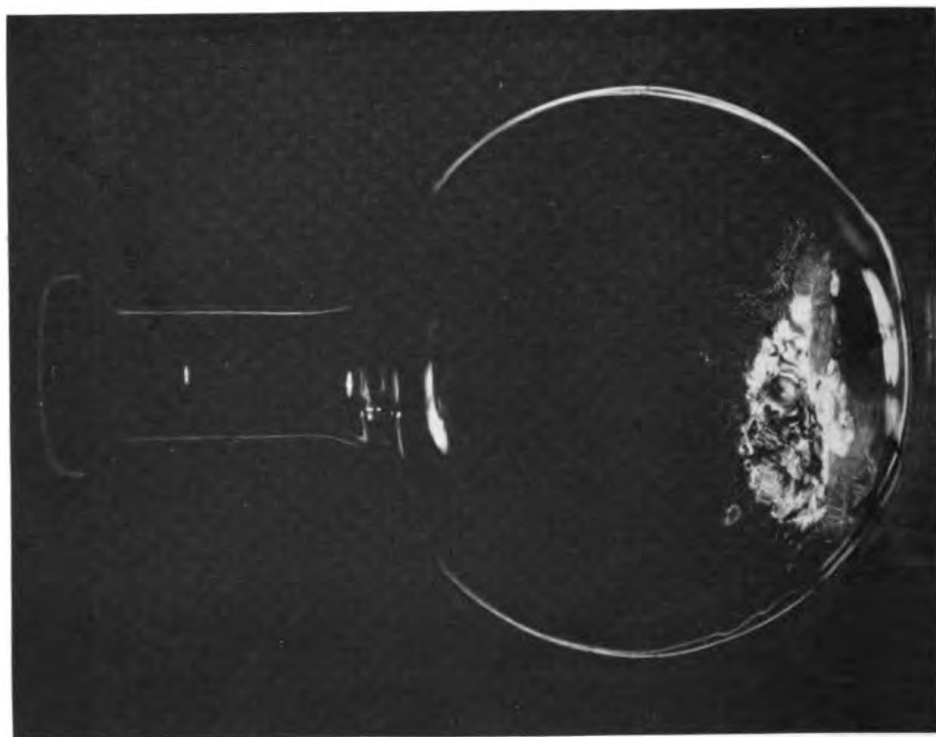


FIGURE 22.—FLASK SHOWING CLOSELY ADHERED BACTERIAL GROWTH.

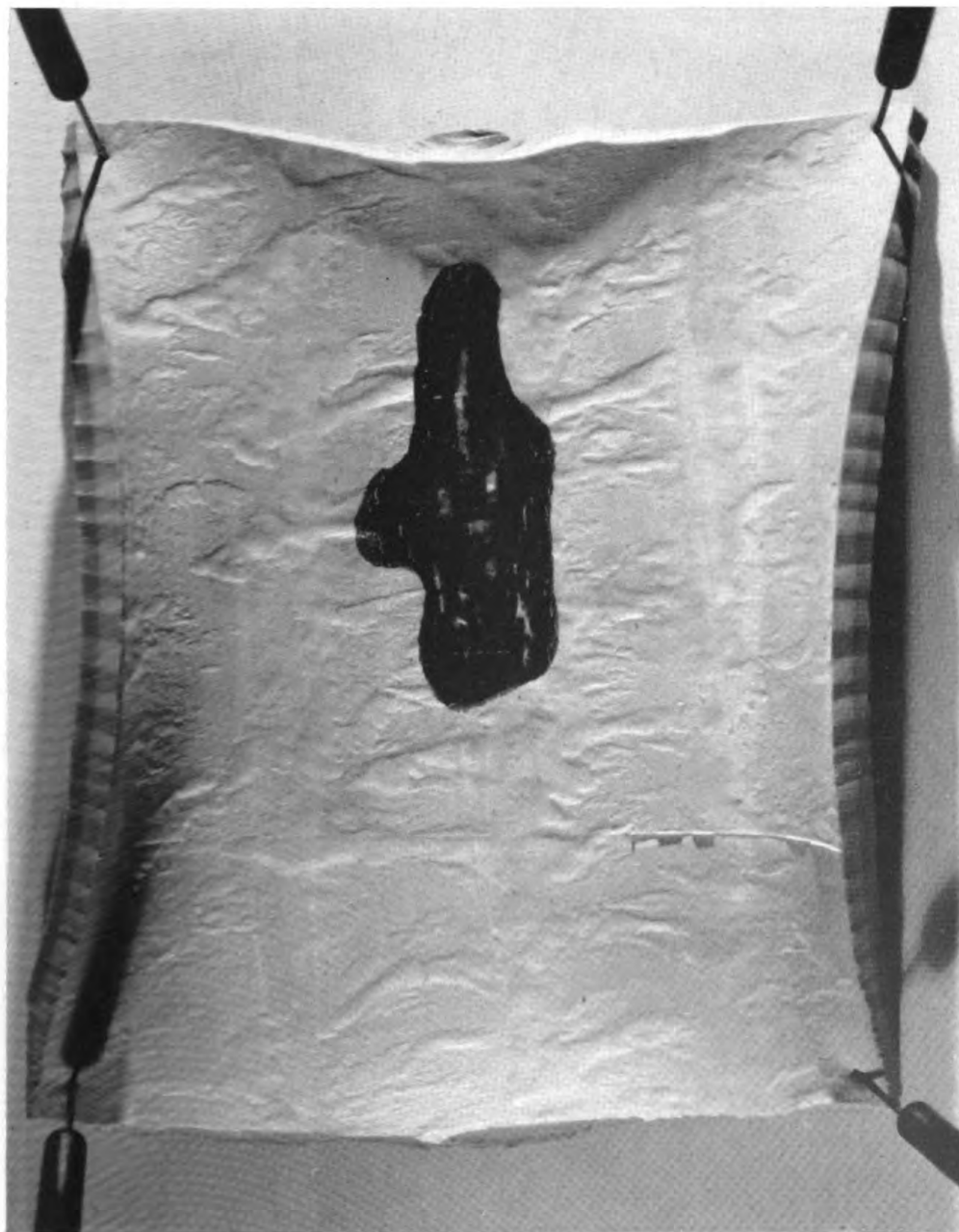


FIGURE 24.—INTERLINING OF TUBE SHOWING RESIDUE.

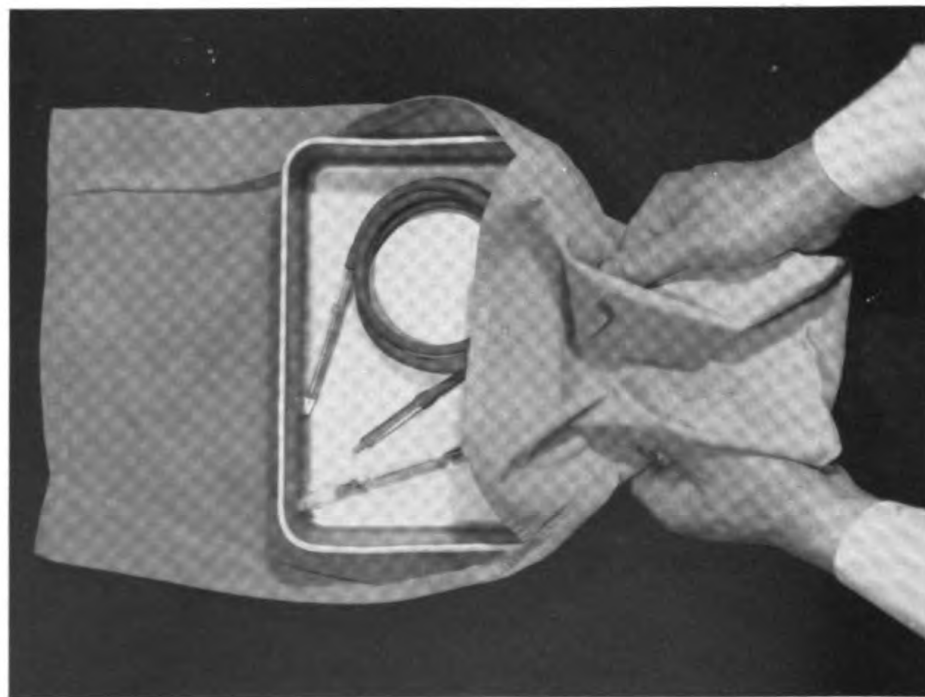


FIGURE 25.—INFUSION APPARATUS AND STERILIZING ENVELOPE.

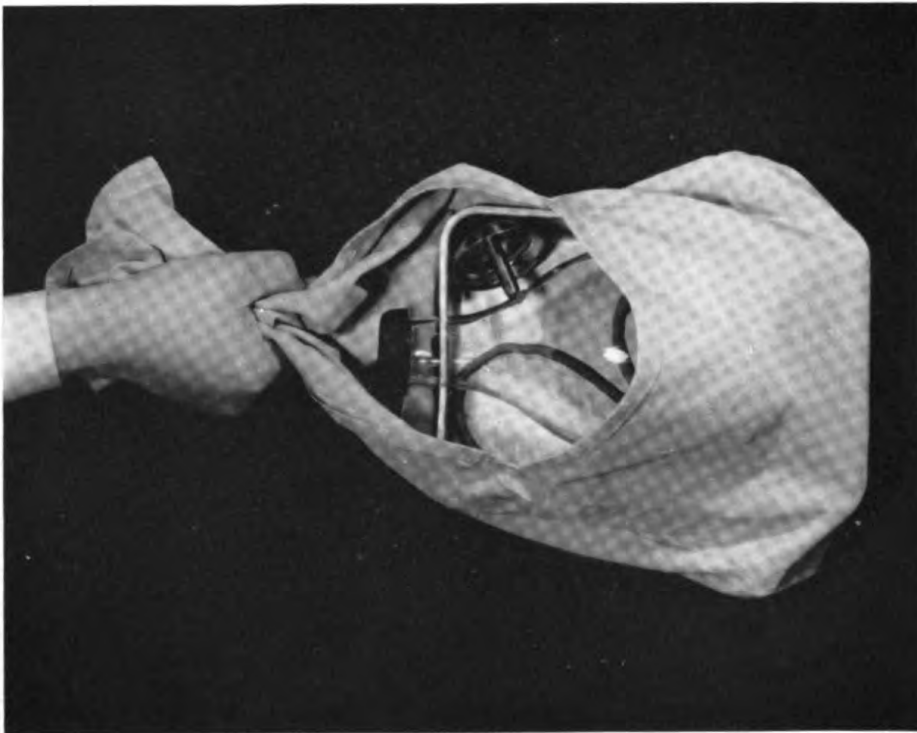


FIGURE 26.—ENVELOPE USED TO CARRY NONSTERILE EQUIPMENT.

be sterilized with stilets in place because the electrolytic action set up between the stilet and the needle causes early corrosion and weakening.

The infusion apparatus is reassembled without drying and arranged in a clean aluminum tray. This tray is inserted into the sterilizing envelope, with the inner flap tucked beneath the pan and the outer flap securely pinned. It is easily extruded by forcing the sides of the envelope together behind the tray (fig. 25). The envelope also serves as a receptacle in which to carry the nonsterile equipment back to the laboratory (fig. 26). The residual water inside the rubber tubing is vaporized in the sterilizer and provides the moisture essential for sterilization. It is important to have the tubing moist because it is difficult to clear sufficient air from a length of dry coiled rubber tubing to attain sterilizing conditions.

STERILIZATION.—The kits are packed into a dressing sterilizer so that the bottoms of the aluminum trays are in a vertical position. This permits the steam to displace the air in the tray and results in rapid development of sterilizing temperatures. It is advantageous to sterilize them alone or with loosely wrapped rubber goods because little heat is required to bring the kits to sterilizing temperature. They should be sterilized for 30 minutes at 250° F. exhaust line temperature.¹³

After sterilization, the envelopes are dried by leaving the sterilizer door ajar for 15 minutes while the steam pressure is maintained in the jacket of the sterilizer. If the kits are stored with the trays inverted, there is little danger of contamination from dust, and they can be kept until needed.

SOLUTIONS

DEXTROSE AND SALINE

Although the United States Pharmacopeia does not specify dextrose suitable for intravenous use, *U. S. P. XI* or *Chemically Pure* anhydrous grades of chemicals are usually satisfactory. Careless transfer of the chemicals must be avoided, and any which touches the hands or drops upon the balance or table must be discarded.

Dextrose may contain amino acids and carbohydrate condensation products which are denatured by the heat of sterilization and appear as white, flocculent precipitates. Various acid dehydration products of dextrose, formed by side reactions during its manufacture, may also be present.¹³ These compounds are colorless, but on aging degrade to levulinic acid, a dark brown compound. This conversion occurs rapidly in hot aqueous solutions or on exposure to light and accounts

¹³ Walter, C. W.: Aseptic technic; sterilization of dressings and dry goods. *Int. Abst. Surg.*, 71: 414, November 1940.

for the yellow color (often mistaken for caramelization) developed during sterilization or storage of some dextrose solutions.

The acceptance of a particular lot of chemical also depends upon the quantity of particulate matter contaminating it. Excessive dust clogs the filters quickly and is evidence of careless handling prior to packaging. The finding of a shoelace tip or a dried insect indicates contamination of a degree likely to introduce foreign proteins into the chemicals. Dirty chemicals must be rejected or purified by adsorptive filtration.

PREPARATION.—Mass filtration and bulk dilution of solutions will be found inconvenient in most hospitals. The problems entailed in handling such large quantities of fluid can be avoided by a technic which also limits potential spoilage due to improper mixing, faulty filtration, careless contamination or uncleanness, to individual flasks. Such a technic ¹⁴ is based upon the filtration of a relatively small proportion of the final solution in the form of a concentrate. This concentrate is then diluted with pure distilled water. In the average hospital, the concentrate is filtered most advantageously into a large volumetric burette whence a suitable portion is measured into a container and distilled water added until the proper dilution by weight is made.

For example, 5 percent dextrose solution is prepared from a fresh stock solution made by adding hot distilled water to 1,000 grams chemically pure dextrose, previously weighed out in a counterbalanced flask, up to a net weight of 2,355 grams. The flask is stoppered with a clean rubber stopper and shaken until solution is complete. This 50 percent stock solution is then filtered through a porcelain filter (F. G.-450-10) or adsorptive filter (Seitz Uhlenhuth 60 mm. Germicide EK), with the aid of suction, directly into a pyrex burette. The filtrate should be crystal clear and colorless. The stock solution can be decolorized and clarified if necessary by adding 1 to 2 percent activated charcoal (Norit—decolorizing carbon—P1731, Research Laboratory, Eastman Kodak, Rochester, New York) prior to filtration. One hundred cubic centimeters of the filtrate are measured into a counterbalanced container, and distilled water is added to a net weight of 1,066 grams.

STERILIZATION.—Solutions of sodium chloride and dextrose are sterilized immediately in an autoclave at 250° F. (exhaust line temperature) for 30 minutes. After sterilization the steam supply to the autoclave is shut off to permit the autoclave to cool to 200° F. before it is opened. In this way concentration of solution resulting from the ebullition of steam following sudden relief of pressure is avoided. If the flasks are hermetically sealed, they can be stored indefinitely without impairing their value as safe parenteral fluids.

¹⁴ Walter, C. W. Preparation of safe intravenous solutions. *Surg. Gynec. & Obst.* 63: 5, 643, Nov. 1936

PROCAINE HYDROCHLORIDE

Solutions of procaine hydrochloride for local anesthesia must be isotonic to avoid damage to cells¹⁵ and tissue necrosis with consequent postoperative induration. For full anesthetic effect,¹⁶ procaine must be alkaline, as the acid solution has but slight action.¹⁷ Alkaline solutions deteriorate so rapidly that they cannot be sterilized or stored. Acid solutions, however, withstand sterilization well^{18 19} and can be stored indefinitely in hermetically sealed containers of hard glass. Procaine solutions should be crystal clear and colorless. The drug forms a white flocculent precipitate or collects as oily droplets at the surface at a pH greater than 8. Straw-colored or brownish tints are due to degradation²⁰ to p-aminobenzoic acid and diethylaminoethanol. Such solutions irritate tissues and have lost their maximum anesthetic effect.

Although procaine must be sterilized and stored in acid solution and administered as an alkaline solution, stable procaine solutions with full anesthetic effect can readily be provided in every operating room. Two solutions are necessary. A 1 percent solution made isotonic with sodium chloride serves as an ideal infiltration anesthetic even though it has an acid pH for the tissue fluids possess sufficient buffer to shift the reaction of the injected fluid to an alkaline pH. Hence the anesthetic property is developed in the tissues themselves.²¹ Unfortunately, large nerves do not contain sufficient tissue fluid to exert this buffer action, and alkaline novocaine must be injected.¹⁷ This can easily be done by adding the alkali immediately before injection, just as many surgeons add epinephrine hydrochloride solution (1 : 50,000) to delay absorption of the anesthetic and to prolong its action three or more times.²²

PREPARATION.—To avoid an alkaline pH at the solution-glass interface, hard glass containers (Corning Glass Works: "Boston Rounds"—Catalog No. 650.) must be used. If these are hermetically sealed, the novocaine can be stored indefinitely with but minor loss (2 percent during 8 years).¹⁹ The bottles must be thoroughly cleaned, rinsed with pyrogen free distilled water, and inverted to drain dry.

The 1 percent solution (pH 2.9) is made by dissolving 10 grams of procaine hydrochloride and 7.5 grams of C. P. sodium chloride to a

¹⁵ Gilman, S.: Treatment of dangerous reactions to novocaine. *New Eng. J. Med.* 219: 841, November 1938.

¹⁶ Gros, O.: Ueber de Narkotika und Lokalanästhetika. *Arch. f. Exper. Path. u. Pharm.* 63: 80, 1910.

¹⁷ Gerlough, T. D.: Influence of pH on activity of certain local anesthetics as measured by the rabbits' cornea method. *J. Pharmacol. & Exper. Therap.* 41: 307, March 1931.

¹⁸ Barlow, O. W.: Winthrop Chemical Co., Inc.: personal communication, October 1, 1940.

¹⁹ Schou, S. A. and Abildgaard, Jens: Decomposition of novocaine solutions upon sterilization and storage. *Pharma. Acta Helv.* 10: 38-47, 1935. Abstracted by S. Waldbott in *Chemical Abstracts*, vol. 29, pt. III, p. 8236, 1935.

²⁰ Winthrop Chemical Co., Inc., Rensselaer, New York: personal communication.

²¹ DeTakata, G.: Preparation of local anesthetic solutions. *Bull. Am. Coll. Surg.* 17: 40, December 1933.

²² Bieler, R. N.: Applied pharmacology of local anesthetics. *Am J. Surg.* 24: 500, December 1936.

liter of boiling hot freshly distilled water containing 0.1 cc. of 10 N hydrochloric acid. After solution is complete 50 cc. quantities are transferred to clean bottles. If screw cap bottles are used, the caps are started on but not turned down tightly.

The alkaline solution is made by diluting 1 cc. of 10 N sodium hydroxide to 100 cc. with freshly distilled water. This solution is transferred into clean 20 cc. serum bottles²³ with the aid of a syringe and a 16-gauge needle. The bottles are stoppered with rubber stoppers and as much air as possible is aspirated from them through a fine needle which is affixed to a 5 cc. syringe and inserted through the stopper. Evacuation of the air prevents the stoppers from being blown out during sterilization.

STERILIZATION.—The bottles are sterilized by exposure to saturated steam for 20 minutes after the temperature in the exhaust line has reached 250° F. After the sterilizer has cooled, the screw caps are tightened—hermetically sealing the bottles.

USE.—For infiltration anesthesia the solution can be used as it comes from the bottle. For nerve block 1.5 cc. 0.1 N of the sodium hydroxide are aspirated from the vial with a syringe and added to a full bottle (50 cc.) of novocaine to render it alkaline and anesthetic. For spinal anesthesia the best solvent for procaine is the patient's own spinal fluid which buffers the drug before it is injected intrathecally.

MORPHINE SULFATE

To have morphine available for instant injection it is convenient to provide it in sterile solution in rubber stoppered vials whence it can be quickly withdrawn into a sterile syringe.

Like procaine, morphine is unstable in alkaline solution; accordingly it must be made up with hot freshly distilled water and kept in hard glass containers. Properly sterilized and sealed morphine solutions can be stored for several months without appreciable loss of narcotic action. Satisfactory formulae for solutions of morphine are:

Morphine hydrochloride.....	0.10 gm. ²⁴
Dilute hydrochloric acid, U. S. P.....	0.05 cc.
Distilled water to.....	10.00 cc.
or	
Morphine hydrochloride.....	1.0 gm. ²⁵
Para-chloro-meta-cresol.....	0.1 gm.
Distilled water to.....	100.0 cc.

The solutions are bottled in 20 cc. hard-glass serum bottles. The bottles are stoppered with rubber serum stoppers, the residual air

²³ Kimble Glass Company, Vineland, N. J., Catalog No. 15085, bottles, Catalog No. 1920, stoppers.

²⁴ Morphine in solution. J. A. M. A. 105 : 1456, November 1935.

²⁵ Davis, H.: Preparation and preservation of morphine injections. Quart. J. Pharm. & Pharmacology, 8: 4, 683, 1935.

evacuated through a fine needle, and sterilized by exposure to saturated steam at 250° F. for 30 minutes.

One cubic centimeter of either solution contains 10 mg. of morphine hydrochloride. If sterile 1.5 cc. syringes are provided with the bottles of solution, the danger of accidental overdosage is minimized.

Atropine sulfate cannot be preserved in solution because it deteriorates rapidly.²⁶ Where a solution containing both atropine and morphine is desired for preanesthesia medication, it may be prepared by the following formula:

Morphine HCl.....	1.0 gms.
Atropin Sulfate.....	0.006 gm.
Dilute HCl U. S. P.....	0.05 cc.
Distilled water to.....	10.00 cc.

This solution is sterilized as above but must be used within 24 hours.

ARSENICALS

The preparation of the various arsenicals for intravenous injection requires sterile, pyrogen-free distilled water and apparatus. These essentials can be controlled as previously described. Instructions for the actual preparation of the solutions accompany every ampoule of arsenical and should be followed in detail.

SULFANILAMIDE AND SULFAPYRIDINE

Aqueous or saline solutions of these drugs for parenteral administration should be prepared as described under the section dealing with the preparation of dextrose solutions. They can be prepared as 0.5 percent solutions by adding 5 grams of the crystalline drug to a liter of 0.85 percent saline. Pyrogen-free distilled water and apparatus are imperative because these drugs are usually administered to critically ill patients for whom an untoward reaction might be serious. The solutions are sterilized by exposure to saturated steam at 250° F. for 30 minutes.²⁰

When the crystalline drug is applied topically, the crystals should be taken from a freshly opened package with aseptic precautions. The crystals are sterile when packaged.²⁰ If such use is routine, a supply of the drug, packaged for this purpose, can be obtained, thereby eliminating chance contamination.

SODIUM TETRAIODOPHENOLPHTHALEIN

Alkaline solutions of this drug are relatively stable when protected against absorption of carbon dioxide. The drug precipitates from acid solutions, while an alkaline solution is clear and purplish red.

A suitable solution for intravenous injection is prepared by dissolving 2.5 grams in 28 cc. of hot, freshly distilled water in a clean

²⁶ Rapid loss of pharmacologic power of atropin. J. A. M. A. 64: 705, 1915.

bottle. Shaking or cooling hasten the absorption of carbon dioxide and must be avoided. The screw cap is put on loosely and the bottle is sterilized as described for procaine. Sterile, hermetically sealed solutions can be stored indefinitely. Bottles containing sodium tetraiodophenolphthalein should not be shaken prior to use as this stirs up the sediment, which settles out of unfiltered solutions as they cool. Solutions in which a gross precipitate appears should be discarded.

SODIUM CITRATE

Three percent solution of sodium citrate is prepared by dissolving 30 grams of C. P. sodium citrate in a liter of freshly distilled water. This solution is filtered through a porcelain filter into a large burette whence 75 cc. quantities are measured into clean containers. It is sterilized for 20 minutes at an exhaust line temperature of 250° F. and when hermetically sealed (fig. 27) can be stored indefinitely. Such solutions are instantly ready for collecting blood simply by removing the stainless steel stopper and inserting the glass side arm tube (fig. 28). The blood flows (by the aid of venous pressure and gravity) through the needle and short rubber tubing into the stainless steel tube whence it dribbles into the citrate which is gently swirled (fig. 29). The blood can be infused from the same container by substituting a longer stainless steel tube for the donor's tube (fig. 30) and inverting the flask. The blood runs through narrow saw cuts in the glass side arm tube into the annulus about the steel tube and then out the side arm to the recipient's tube. The longer steel tube serves as a vent to relieve negative pressure as the flask is emptied.

CONCLUSION

Knowledge of the cause of untoward reactions and the intelligent application of the principles described can do much to assure safe infusions and transfusions. Cholecystography can be made uneventful, local anesthesia effective, and parenteral therapy successful by the observance of this simple technic for the preparation of appropriate solutions.

NONINDUSTRIAL EYE INJURIES¹

TREATMENT, WITH ILLUSTRATIVE CASES

By Commander Warren D. Horner, Medical Corps, United States Naval Reserve

San Francisco Municipal Hospital contains 1,400 beds and serves a community of more than 600,000 souls. It is the center of an efficient emergency hospital system which embraces 7 branch hospitals located in various sections of the city. The visiting and resident staffs in

¹ From the division of ophthalmology, department of surgery, University of California Medical School. Presented before the Pacific Coast Oto-Ophthalmological Society, Spokane, Washington, June 26, 1940.

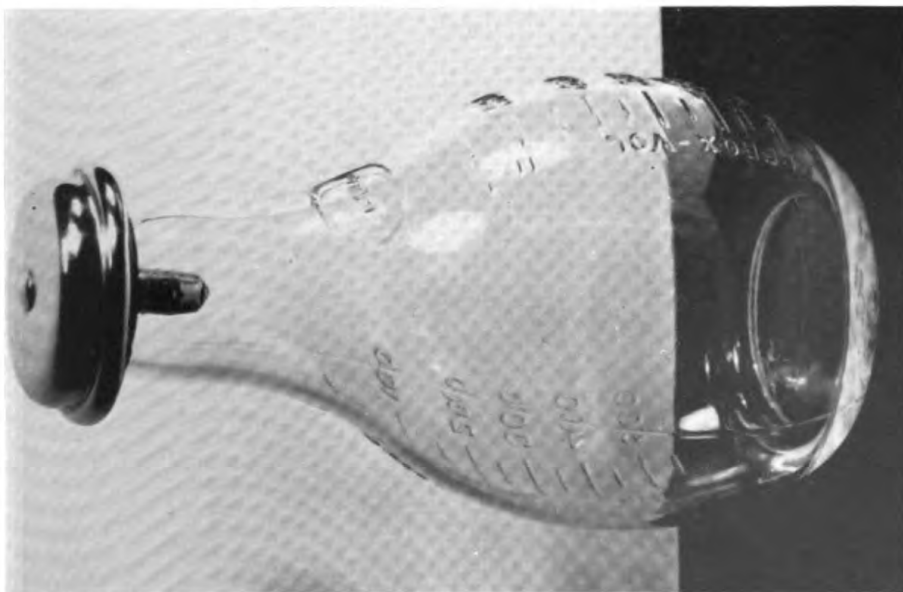


FIGURE 27.—HERMETICALLY SEALED FLASK.

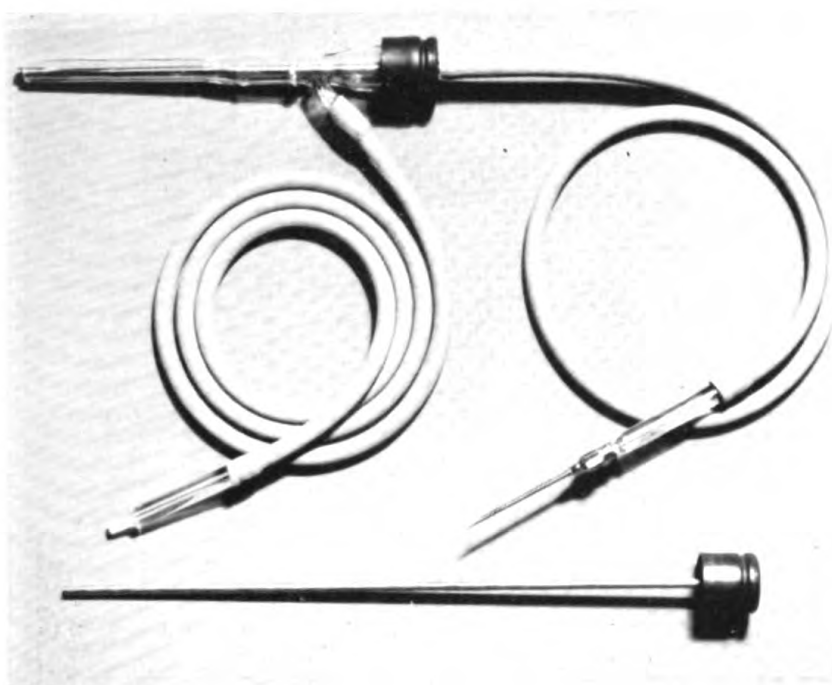


FIGURE 28.—ILLUSTRATION SHOWING STAINLESS STEEL STOPPER AND GLASS SIDE ARM TUBE.

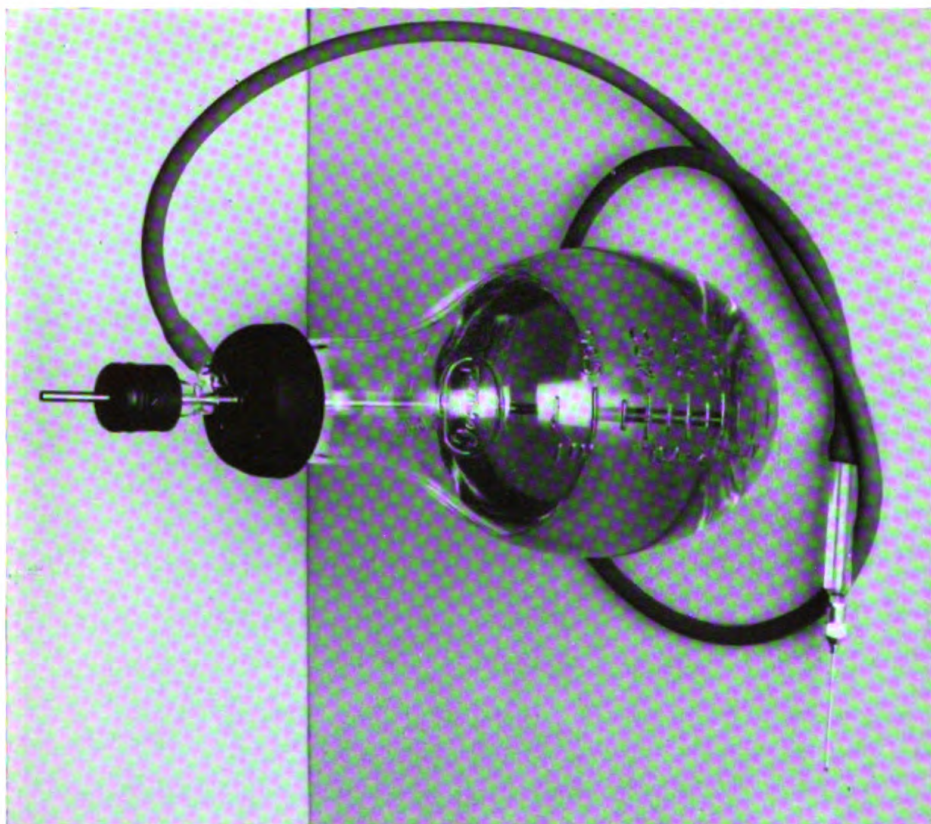


FIGURE 30.—ILLUSTRATION SHOWING METHOD OF GIVING BLOOD TO THE RECIPIENT BY INVERTING FLASK.



FIGURE 29.—ILLUSTRATION SHOWING FLASK AND TUBES FOR COLLECTING BLOOD BY THE AID OF VENOUS PRESSURE AND GRAVITY.

ophthalmology thus have the opportunity of caring for a wide variety of primary and secondary injuries to the eye. The title of non-industrial eye injuries was chosen purposely so as to include traffic accidents, household injuries, and a large group intimately connected with alcoholism and other misconduct, namely, fights, falls, and robberies.

Slang parlance includes the cryptic expression "beaten up." I would like to elaborate upon the practical meaning of this phrase. "Beaten up" ranges from murder, down through the various degrees of brutality. It includes blows from fists, lead pipe, sand bags, bottles, brass knuckles, clubs, kicks, knives, and forcible ejection from moving automobiles. No part of the human anatomy is immune, hence there result fractured skulls and other bones, broken noses, ruptured globes, ruptures of viscera, cuts, lacerations, and contusions *ad infinatum*.

Periorbital injuries are quite common, usually in the form of extensive ecchymosis and swelling of the lids or cuts along the eyebrow. The extent of injury must be determined at the first examination, so it must be thorough and should include vision, inspection of the cornea, globe, fundus, and intraocular tension by fingers. Lid retractors and conjunctival anesthesia may be necessary for this. Subcutaneous emphysema of the face or orbit following a fracture of the nose is occasionally seen. A fair percentage of wounds about the eyebrow become infected and must be opened and drained. I recently saw a neglected, infected wound of the upper eyelid in an alcoholic who had fallen downstairs. The skin of the lid had sloughed and retracted so much that the lid was completely bare. The levator muscle was intact. The lid margin was lifted and sutured to the retracted skin with considerable difficulty. The result resembled a ptosis operation with temporary lagophthalmos which persisted for nearly a week.

Incised and lacerated wounds of the lids are fairly common and are frequently extensive. In their repair one must be guided by the principles of fresh wound healing which will briefly be reviewed.^{2 3 4 5}

PRINCIPLES OF FRESH WOUND HEALING

HAEMOSTASIS.—The ideal control of hemorrhage neither permits the development of hematoma nor justifies the use of unnecessary or mass ligatures, nor dressings tight enough to starve injured tissues. Hemorrhage in a wound acts as a foreign body and should be removed because it distends living tissue, hinders the blood supply of cells and encourages the growth of bacteria.

² Whipple, A. O.: Essential principles in clean wound healing. *Surg. Gynec. & Obst.* 70: 257, February 1940.

³ Reid, M. R. and Stevenson, J.: Treatment of fresh wounds. *Internat. Abs. Surg.* 66: 313, April 1938.

⁴ O'Connor, G. B.: Immediate management of surface injuries. (Presented before California State Med. Assoc. Meeting, Coronado, May 1940.)

⁵ Smelo, L. S. Problem of wound healing. *Arch. Surg.* 33: 493, September 1936.

INFECTION.—Carefully cleanse and sterilize skin surfaces. Use strict aseptic technic in the repair. Fresh wounds prior to the lapse of 6 to 8 hours may contain bacteria as a surface contamination, but these have not yet invaded living tissues. Efforts to kill bacteria in a fresh wound may not only be unsuccessful but may do unwarranted harm to living cells. Bacteria are thus best removed by thorough irrigation with normal salt solution. Bacteria feed upon dead or devitalized cells, hence damaged tissue should be cut away to prevent necrosis. Normal tissue, if damaged by rough handling, improper ligatures and sutures, or strong antiseptics will become dead tissue.

SEVERED TISSUES should be replaced and maintained in apposition to simulate normal, anatomic relations as far as possible.

NUTRITION of the wounded tissues should be restored and conserved. Preserve blood supply; avoid mass ligatures; use fine hemostats, needles, and ligatures; tie sutures loosely to allow for swelling; avoid tension in sutures.

REST.—The patient and the repaired wound should be placed at rest, with due attention to the general comfort of the patient and to the support or immobilization of the wounded part.

To be more specific, we have found the following technic satisfactory:

TREATMENT TECHNIC

Cleanse skin thoroughly with soap and sterile water, clip lashes and shave eyebrow if involved. Follow by tincture of merthiolate. Irrigate wound with warm sterile saline or if wound is dirty use equal parts of saline and peroxide of hydrogen in an ear syringe. This not only removes foreign matter and blood clots, but also acts as a hemostatic. Use chromic cat gut, double or triple O for buried sutures. Use silk in the conjunctiva and skin, taking care not to make the sutures too tight, nor the flaps under too much tension.

Where an eyelid had been torn at the inner canthus, or the skin has been beveled like a harness maker's splice, shrinkage occurs so that one may be at a loss to know just where to reattach the torn flap. A simple landmark is offered by the lacrimal puncta which are normally in a vertical line so that one may serve as a reference point for the other.

Where a canaliculus has been lacerated or cut across, Spaeth⁶ advises its immediate repair using a No. 2 Bowman probe as a core, which is inserted into the uncut end of the canaliculus *via* the lacrimal punctum. The lacerated canaliculus is closed by 3 untied sutures over the probe which is gently passed through the sac into the nasolacrimal duct. The probe is then bent down to a right angle at the sac to conform to the horizontal course of the canaliculus. The protruding end of the probe is next cut off beyond the canaliculus and

⁶ Spaeth, E. P.: Principles and Practice of Ophthalmic Surgery, Lea & Febiger, Philadelphia, 1939.

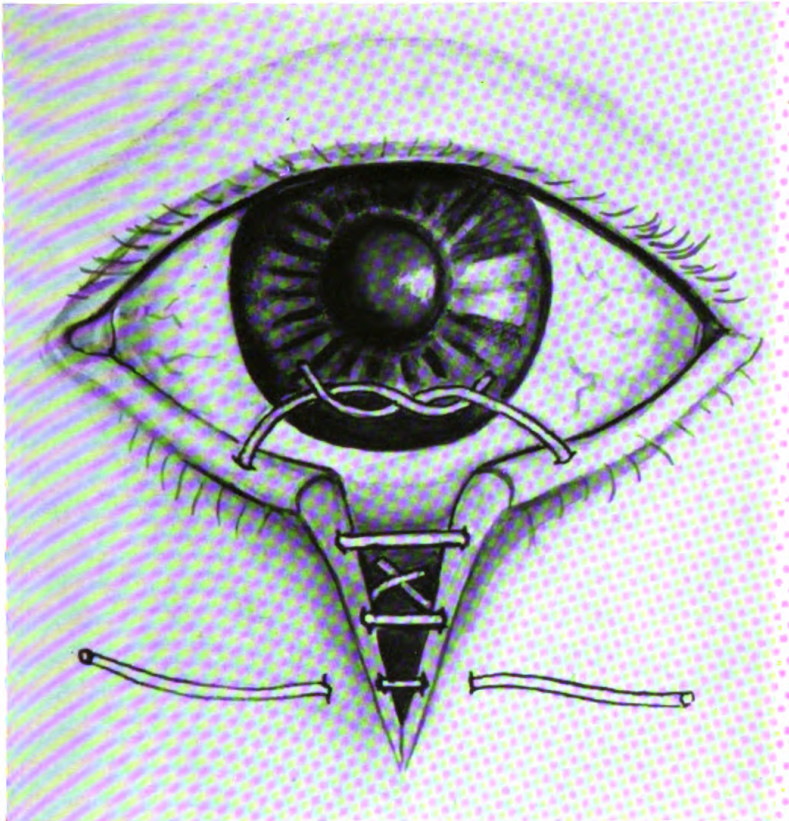


FIGURE 31.—CLOSURE OF VERTICAL CUT OF LID MARGIN.

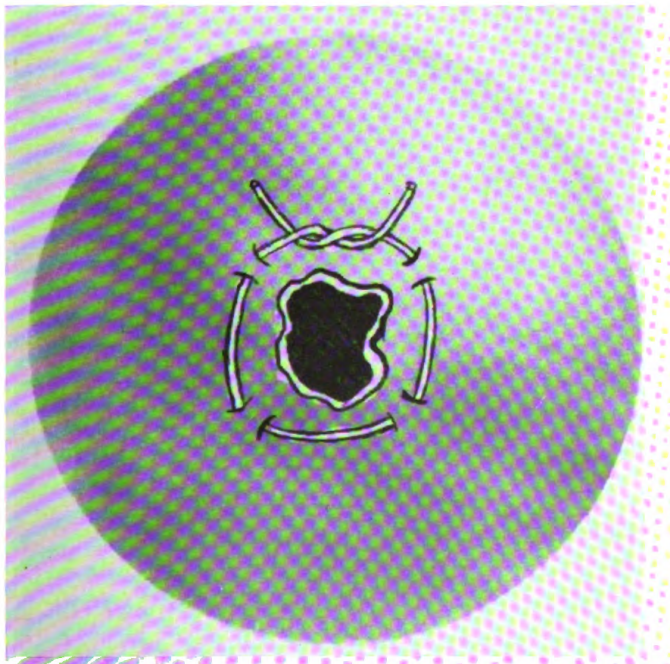
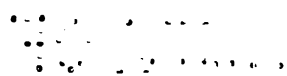


FIGURE 33.—PURSE-STRING CONJUNCTIVAL SUTURE.



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bent out of the way below the lid margin. The three sutures are now tied over the self-retaining probe which is allowed to remain in situ for 6 days after which it and the sutures are carefully removed.

Vertical cuts of the lid margins are repaired with silk sutures in the conjunctival surface which are tied first, silk in the skin, and a finishing suture of silk in the free margin of the lid which bridges the gape. (fig. 31). This suture should be placed about 3 mm. from the edges of the defect so that it will not cut out of the friable tarsus. It is well to cross the threads as a trial before tying this suture, so that one can make sure that it will close the defect and leave neither a notch nor a step in the lid margin. Binocular dressings are, of course, essential.

An excellent double mattress suture for the repair of cuts through thinskin areas has been demonstrated to me by Dr. Edmund Butler Chief Surgeon of our Emergency System (fig. 32).

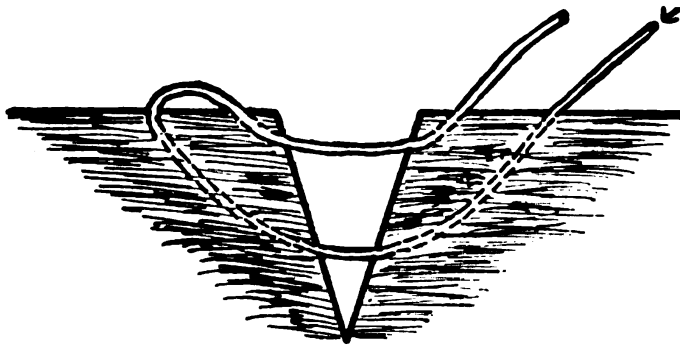


FIGURE 32.—Double mattress suture.

Perforating wounds of the orbit in our experience have resulted from gun-shot wounds, automobile glass, and stab wounds. In one case a jagged piece of automobile glass the size of a 25-cent piece cut through the upper lid and lodged in the orbit behind the eyeball where it severed the optic nerve. It could not be seen and was only discovered in probing the wound. The possibility of a retained foreign body in such cases should always be borne in mind.

In another case a Mexican woman was stabbed thru the base of the upper lid. The knife penetrated the eyeball behind the equator, necessitating later enucleation. None of the perforations into the orbit which I have seen developed orbital abscesses.

Corneal injuries are common and are often neglected so that the patient enters the hospital with a serpigenuous ulcer or corneal abscess. Opening the cornea by a thin Graefe knife after the manner described by Harold Gifford⁷ and called by him "delimiting keratotomy" may save enough clear cornea for vision or at least to permit a later optical iridectomy. The cut is made through the cornea tangential to the ulcer at its advancing edge and with good illumination is not difficult. As soon as the cornea is penetrated, the knife is held parallel to the

⁷ Gifford, S. R.: Handbook of Ocular Therapeutics. Lea & Febiger, Philadelphia, 1932.

iris so that the lens is not injured. We have always used this procedure in combination with repeated doses of intravenous typhoid vaccine. The corneal wound may be reopened several times a day to allow free drainage of the aqueous. In one case, a second incision at right angles to the first was later necessary in order to preserve a clear corneal quadrant.

Contusions of the globe from fights, air-rifle shot, falls, firecrackers, whiplashes, handballs, tennis, and base balls are fairly common. Hemorrhage into the anterior chamber (or vitreous) is the usual sequel. While this ordinarily absorbs without incident, there is always danger of recurrent or post-traumatic hemorrhage as has been emphasized by Cordes and Horner in 1932.⁸ This may occur at any time from the second to the fifth day and is far more dangerous than the primary bleeding, sometimes leading to secondary glaucoma, or to vitreous hemorrhage which requires months to clear. Such patients should be given bed rest until the danger period has passed. It is not sufficient to tell the patient to go home and "take it easy," for they usually won't do it, as was stressed in the article referred to. Bed rest will not prevent every post-traumatic hemorrhage, but will lessen its occurrence. Moccasin snake venom might be helpful as a prophylactic in such cases. Less frequent complications from contusions include paralytic mydriasis, iridodialysis, traumatic cataract, lens subluxation, ruptures of the choroid, Berlin's opacity and retinal detachment. I recently examined a man who was struck upon the head by a hinged window which suddenly blew shut. While he did not remember being struck in the eye, the lens showed a Vossius pigment ring and there was an extensive Berlin's opacity of the retina.

Perforating wounds of the cornea and sclera have occurred from varied causes such as glass splinters from automobile windshields and broken spectacles,⁹ home workshop accidents particularly in children who have their heads too close to an adult working at a bench. The sudden slip of a screwdriver or a flying metallic fragment may blind the onlooker or the workman. Two small boys received eye injuries from striking loaded rifle cartridges with a hammer just to see what would happen. One lost his eye from a piece of brass from a .30-30 shell which penetrated the globe and lodged deep in the orbit. The other was fortunate in that a fragment from a .22 shell cut the sclera but remained outside the eyeball. One child hammered a giant powder cap and received copper fragments in each eye which, fortunately, absorbed in time, leaving one normal eye and a copper cataract (chalcosis) in the other. This case, which has been reported

⁸ Cordes, F. C., and Horner, W. D.: Contusion of the eyeball with delayed intraocular hemorrhage. *Amer. Journ. Ophth.* 15: 945, October 1932.

⁹ Horner, W. D.: Penetrating injuries of the globe from spectacle glass. *Arch. Ophth.* 22: 439, September 1939.

by Cordes and Harrington,¹⁰ is remarkable in another particular. On admission she maintained that she had hurt only the left eye. However, the fellow eye which looked all right externally was found by ophthalmoscope to contain a bright bit of copper in the vitreous resembling a piece of ferrule from a lead pencil. One should, therefore, assume in explosion cases that both eyes are injured until examination and x-ray pictures prove the reverse. A man chopping wood was struck in the eye by a splinter which penetrated the iris and the lens at its periphery. He pulled out the splinter which was hanging from the eye but a tiny fragment remained in the lens. The anterior capsule sealed the wound. In spite of a violent iridocyclitis, he finally recovered normal vision. The peripheral lens opacity did not increase.

RATIONALE FOR REPAIR OF CORNEAL AND SCLERAL WOUNDS

(1) First consider the possibility of an intra-ocular foreign body. If such exists and is likely to be magnetic, have localizing x-rays taken immediately before repairing the wound. In an emergency the magnet may be applied at once, although localization is a great help.

(2) If likely to be a nonmagnetic foreign body, repair the wound and x-ray for record afterward.

(3) Examine the eye carefully to determine the extent of injury and whether surgical repair is necessary, or possible.

(4) If so, remove to operating room and under suitable anesthesia, cleanse eye and plan repair according to findings. Small wounds may be covered by a suitable flap of conjunctiva after abscising prolapsed tissues. In doing this, prepare the flap and place sutures ready for tying before disturbing prolapsed iris or vitreous. The fibrinous adhesions which have formed in the lips of the wound are usually strong enough to permit the dissection of a flap without danger of further prolapse. When the flap is in readiness, separate the adhesions at the wound edges with a spatula, grasp and abscise the prolapsed tissues and tie sutures immediately. The shallow injection of sterile saline solution or novocain beneath the conjunctiva in the form of a bleb, simplifies a flap dissection and makes it thinner. If the injection is started some distance away from the limbus, the bleb will separate the conjunctiva right up to the limbus where the incision is to be. If the conjunctiva at the limbus is well dissected, it can be drawn down to temporarily cover about 60 percent of the cornea and will return to the limbus when the stitches holding it are removed. This, in my opinion, is superior to a completely isolated bridge flap which must be cut off later. Larger wounds or wounds that gape may require sutures by Kalt needles which are very fine and can be

¹⁰ Cordes, F. C. and Harrington, D. O.: Bilateral absorption of intra-ocular copper with chalcoisis in one eye. *Amer. Journ. Ophth.* 18: 343, April 1935.

introduced into cornea, episclera, or sclera without too much pressure. Such fine silk sutures in no wise prevent use of a conjunctival flap as additional protection and blood supply to the cornea, since they can be removed after the flap retracts. In extensive corneal wounds, I favor a purse-string suture in the conjunctiva which, after dissection from the limbus, may be drawn completely over the cornea. This moulds and supports the cornea so that it is in its proper shape for healing. This same suture can be used in healing indolent corneal ulcers (fig. 33).

Intravenous typhoid vaccine should be given routinely after every penetrating ocular injury and can be repeated daily or every other day as required. Its use in ocular injuries and infections is so well grounded that no further comment is necessary.

In eyes which have been penetrated by spectacle or laboratory glass, the anterior chamber is usually filled with blood so that one must depend upon the x-ray to show whether any glass particles have been retained. In some experiments done last year ⁹ I demonstrated that the routine film of an eye localizer will show a glass spicule as large as $1\frac{1}{2}$ by $1\frac{1}{2}$ by 2 mm. Minute foreign bodies, particularly glass or wood in the anterior portion of the eye, may also be accurately demonstrated by the skeleton-free method of Vogt.¹¹ This technic utilizes a dental film placed nasally to the eyeball and pressed as deeply as possible into the orbit. Soft roentgen rays directed from the temporal side will reach the film through the anterior eyeball without interference by the bones of the orbit.

Out of seven cases of scleral ruptures, five resulted from fights, one from an alcoholic collision with the edge of a piano and one from being beaten over the head with the heel of a woman's shoe. The latter makes a vicious weapon as judged by its effect upon the eye and scalp of this injured man. The majority were indirect ruptures occurring in the upper inner quadrant of the globe adjacent to the prominence of the superior oblique pulley. Scleral rupture from being hooked by a cow's horn was not observed, although this is prominently mentioned as a statistical cause. Six of the injured eyes necessitated enucleation eventually. The seventh is still under observation. The lens in this case lay under the conjunctiva on admission. One eye could not be immediately removed on account of the poor general condition of the patient. A scleral staphyloma developed within 4 weeks. It is noteworthy to mention that the scleral rupture in every case was always more extensive when it was exposed at operation than it appeared to be clinically.

¹¹ Vogt—cited by Berens, C. in *The Eye and Its Diseases*. W. B. Saunders Co., Philadelphia, 1936, p. 936.

OPTIC NERVE INJURY

Damage to the optic nerve following head injuries has been seen occasionally. Partial or complete blindness may be noted immediately after the injury. Of three cases, one followed a head-long fall from a motor cycle with blindness and later atrophy in one nerve, another followed a kick in the head which caused blindness in a one-eyed individual, while the third resulted in blindness in one eye, partial blindness in the other as the result of a fractured skull from a fall downstairs. In such cases the loss of consensual reflex is an important diagnostic sign of optic-nerve injury.

In discussing the cause of the visual damage ¹² Rea states that the explanation lies not so much in the number of fractures which pass through the optic foramina, but in extravasation of blood into the sheath of the optic nerve. This blood may come from the middle fossa of the skull or from ruptured nutrient vessels lying between the nerve and its coverings or from the central vessels of the retina near the eyeball where they traverse the intervaginal sheath before entering the optic nerve. Hemorrhage into the optic sheath may thus follow slight injuries which cause a sudden change of pressure within the orbit and without intracranial hemorrhage.

Rawlings ¹³ says that the vast majority of anterior fossa fractures avoid the immediate vicinity of the optic foramen and pass by preference between the two foramina or diverge toward the sphenoidal fissure. This contention is supported by Vance ¹⁴ who showed that in only 10 percent did fissured fractures involve the optic foramen. Pringle ¹⁵ examined 174 cases postmortem who died from skull fractures. Of these, only 7.3 percent had fractures involving the optic foramen. Two of these involved fracture of its roof without injury to the optic nerves or hemorrhage into the sheath. Sixteen cases or 9.2 percent showed hemorrhage into the sheath of the nerve.

In contradistinction to anterior fossa fractures, von Hoelder's ¹⁶ 88 cases of fractures of the base involved the orbit in 90 percent and the optic canal in 61 percent.

Statistics thus show a large proportion of optic nerve injuries unexplained by either hemorrhage into the sheath or fractures into the canal. Some of these are undoubtedly due to fracture or displacement of the anterior clinoid processes pressing upon the nerve while others may be due to lacerations, concussion, stretching, and vascular damage to the chiasm as emphasized by Traquair.

¹² Rea, R. L.: *Neuro-Ophthalmology*. C. V. Mosby, St. Louis, 1938, p. 408.

¹³ Rawlings, L. B.: *Head Injuries*, Oxford University Press, 1934.

¹⁴ Vance, B. M.: Fractures of the skull; compensation and causes of death. *Arch. Surg.* 14: 1023, May 1927.

¹⁵ Pringle, J. H.: Atrophy of optic nerve following diffused violence of the skull. *Brit. Med. J.* December 16, 1923, p. 1154.

¹⁶ von Hoelder, quoted by Marzollins, E.: Post-traumatic atrophy of optic nerve. *Inter. Clinics* 3:186, 1918.

Late optic atrophy following head injuries may be due to contusions of the nerve, pressure of callus, hemorrhage, traumatic meningitis, atrophy from traumatic papillitis (rare) and to cisternal arachnoiditis of traumatic origin which may involve the chiasmal region to cause bilateral primary optic atrophy years after injury.¹⁷

INGUINAL HERNIA

INCIDENCE IN CIVIL SERVICE EMPLOYEES AND RELATION TO RELAXED RINGS

By Lieutenant Commander Francis P. Gardner, Medical Corps, United States Navy

The question of the incidence of hernia in civil service employees has brought up several questions, among them: first, how may a hernia be diagnosed, second, what is a relaxed ring, and third, what relation has a relaxed inguinal ring to the incidence of hernia?

Disregarding the complete hernia which is easily diagnosed, what constitutes a hernia? This is a debatable question; some say any relaxed ring with an impulse on straining or coughing is at least a potential hernia. This is not believed to be a fair test, as it does not necessarily prove the existence of a sac, which is essential for diagnosis. In the diagnosis of small hernia a more satisfactory rule, repeatedly confirmed by operation, is that laid down by Commander L. M. Schmidt, Medical Corps, United States Navy.

When the examining finger feels an impulse on coughing and this impulse persists, the patient continuing to cough as the finger is withdrawn, that is to say, the impulse follows the finger, then a sac exists, and abdominal viscera has entered it.

As to the second question, the personal equation of the examining surgeon seems to play a big part; the method of the examination and the interpretation of the findings seem to vary. DaCosta states:

In a healthy man the external ring should admit the tip of the little finger, but not the end of the index finger. If the end of the index finger can be entered into the ring that aperture is dilated, and even if there is no hernia in the canal, in the future a hernia will probably descend.

From the reference Handbook of Medical Sciences, quote:

The external ring varies in size, depending upon the development of the inter-columnar fibers. Normally, the opening will admit the end of the finger, but this is modified by the position of the body. (Note the examining finger not designated.)

Babcock does not define a normal ring.

This variance in findings is substantiated by a 2-year check of records at a navy yard dispensary during which time the medical examiners changed. In 1936 there were 2,203 applicants examined. From this number there were 861 relaxed inguinal rings or 39.07 per cent. In 1937 there were 1,228 applicants examined, from which

¹⁷ Vall, D.: Optochlasmic arachnoiditis. Arch. Ophth. 30: 384, September 1938.

number there were 307 relaxed inguinal rings or 26.62 percent noted; making 12.35 percent difference in findings. It seems that if some definite rule could be adopted and generally used it would be valuable in determining the future of applicants for employment. I have adopted the following rule:

An external ring approximately twice the size of an adult male's index finger with evidence of loss of muscle tone is definitely relaxed.

As to the third question, it is hoped that the following statistics will throw some light on the subject. Since 1931 the records of this dispensary show that there have been 63 hernias diagnosed and of these there is no record of entrance examination in 10 cases as they were employed prior to 1926, before which time no records were kept, which leaves 53 hernias diagnosed that had a record of their examination for employment. Forty-two of these 53 had normal inguinal rings when examined for employment and 11 had relaxed rings.

Since 1926 there have been 24,934 examinations for employment, and from these 24,934 there have developed 53 hernias or 0.212 percent or an incidence of 2.12 per thousand. There were 21,521 applicants with normal rings. From these 21,521 there developed 42 hernias or 0.176 percent or an incidence of 1.76 per thousand. There were in this series 3,413 applicants with relaxed rings, and from these 3,413 there developed 11 hernias or 0.322 percent or an incidence of 3.22 per thousand.

From the foregoing figures the incidence of inguinal hernias in this group of civil employees, comprising third and fourth decades, is 2.12 per thousand, the incidence of hernias in those with normal inguinal rings is 1.76 per thousand, and the incidence of hernias in those with relaxed rings is 3.22 per thousand. Bearing in mind the personal equation of the examining surgeon, the incidence of relaxed rings in this age group is 136.80 per thousand. From these figures the civil employee with relaxed inguinal rings has a 2-to-1 chance of developing an industrial hernia and is therefore a more definite risk to the Government.

Even though weakened muscles and relaxed inguinal rings predispose to the development of hernias it seems to be the opinion of a great many surgeons that hernias do not occur suddenly and that the sac is either congenital or has been forming for years before the trauma to which the hernia is attributed occurs. Though the muscle is weak, unless there is a stretching or pouching of the peritoneum, actual hernia will not develop. It is the opinion of the author that if the rule cited or some other definite method is adopted to detect the presence of such a sac before any evidence of hernia has manifested itself to the employee, a number of small service-connected hernias would be avoided.

ROUTINE KAHN BLOOD REACTIONS

SUPPLEMENTARY REPORT OF 20,000 TESTS MADE ON NAVAL RECRUITS WITH OBSERVATIONS ON THE RELATIONSHIP OF COWPOX VACCINATION TO THE FALSE POSITIVE TEST

By Captain G. E. Thomas, Medical Corps, United States Navy, and Lieutenant (Jr. Gr.) E. W. Garrity, Medical Corps, United States Navy

This report is based on the results of routine blood Kahn tests on every recruit entering the Naval Training Station, San Diego, California, between July 1939 and January 1941. The recruits who come to this station are from Mississippi, Louisiana, and all of the states west of the Mississippi River, except the west North Central States. A preliminary report on the first ten thousand Kahns was made in 1940 and published in the January issue of this Bulletin.

These tests have been run consecutively in the same laboratory by the same technicians. The results have been read as positive, doubtful, or negative. The tests were run in the approved method and results frequently checked by other laboratories, especially that of Kahn at the University of Michigan, and were found to be accurate. When Kahn positive sera were sent to other laboratories, they were found to be Kline and Wassermann positive.

During the first series of 10,000 blood samples, the serum was secured on an average of 12 days after vaccination; in the second series of 10,000, the blood was secured before the men were vaccinated.

In the first series there were 26 men who had false positive tests and 12 men who had persistent positive tests. In the second series, there were 6 men who had false positive tests and 29 who had persistent positive tests. The increase in the persistent positive tests in the second group was believed to be due to the increased volume of recruiting and the lack of time and opportunity for a more thorough investigation prior to enlistment.

In the entire series of 20,000 men there were 73 positive initial Kahn tests of which 32 proved to be false positives before the men left the station, which was usually 2 to 3 months after arrival. Forty-one of the 73 were persistently positive during this period of time. The incidence of the total initial positives was 0.365 percent, the persistent positives amounting to 0.205 percent, and the false positives 0.16 percent. The incidence of false positives was well within the 1-percent allowance for approved laboratories.

The men whose sera gave persistently positive Kahn tests were discharged from the naval service by an order of the Navy Department. Of the 31 men, there were 10 who were definitely believed to have acquired syphilis, 3 who were believed to have congenital lues, 20 who probably had the disease, and 8 that were exceedingly doubtful. These conclusions were based on several complete physical examinations, complete histories with definite statements about previous sores, rashes, other venereal diseases, actual luetic treatments, admissions of

luetie infections, sexual abstinence or promiscuity, especially with prostitutes, sexual contacts with faulty prophylaxis or lack of prophylaxis, statements relative to the parents having or having had the disease, and by persistent positive Kahn tests checked frequently by other precipitation tests and complement-fixation tests. It is interesting to note that 65 percent of these men had promiscuous relations with prostitutes, 28 percent had few sexual contacts, and 7 percent denied sexual intercourse. In addition to the study from the luetic point of view, all other known causes of false positive serology were considered.

The findings suggest a definite relationship between cowpox vaccination and the false positive test. Vaccinations were read as: vaccinia, accelerated (covering all degrees from primary reactions to immune reactions), immune, and failures. Failures were revaccinated until they had some sort of a reaction. In the first series of blood taken after vaccination, there were 26 men who had false positive tests, and of this group 89 percent had either a primary or accelerated reaction; while in the entire 10,000 men, there were only 52 percent who had these reactions. This amounted to 11 percent immunes in the false positive group as contrasted with 48 percent immunes in the entire group of 10,000 men.

In the second group, the blood was taken before cowpox vaccination and, therefore, the vaccination could have had no bearing on the positive blood findings. In this group there were only 6 false positives in contrast to the 26 in the first group. The same technicians performed the tests on the first and second groups.

In the second group of 10,000 men, there was a recruit who had a negative Kahn test on arrival at the station. His first vaccination was a failure and he was revaccinated the following week, subsequently developing a marked vaccinia. About 3 weeks later he was selected as a blood donor for a patient in one of the city hospitals. A Kline test was performed by the hospital laboratory and found to be strongly positive. This was corroborated by a positive Wassermann as well as a positive Kahn test in our own laboratory. This man had not been on liberty since arrival at the station. During the following 3 weeks, the serum was checked by several different tests and by Dr. Kahn's laboratory, as well as the San Diego Naval Hospital laboratory and our own laboratory. The results were found to coincide. The Kahn verification test was reported to be syphilitic in type. Examination on several occasions failed to disclose any physical signs of syphilis, and there was a negative history except for one sexual contact followed by adequate prophylaxis. No sores, rash, or discharge were reported. At the end of 3 weeks, the diagnostic tests became negative and have remained so for over 2 months. It was our opinion that these findings were definitely the result of vaccinia.

Barnard¹ mentions a case which had strongly positive Kahns for 4 weeks following vaccinia. The patient had no evidences of syphilis and the Kahns eventually became negative. He believes that vaccinia was the cause of the positive tests.

We were unable to find any relationship between antityphoid inoculation and the false positive Kahn test. Only 12 percent of the false positive cases mentioned any kind of a reaction. Most of the reactions which we saw were of the 24- to 36-hour variety and there were no blood samples drawn within 46 hours of the antityphoid inoculations, which took place on Saturday mornings. The reactions in the entire group of 10,000 men amounted to 10 percent in contrast to the 12 percent reactions in the false positive Kahn group, which we believed to be of no significant statistical difference. There was no opportunity to search for relationship in the second group of 10,000 because the blood samples were drawn prior to the first inoculation.

There were a few recruits from the Southern Gulf States who developed malaria after arrival at the station. These men had been taking quinine routinely at home, and had their plasmodium infection controlled. When they arrived in San Diego they ceased taking quinine and in a short time manifested clinical malaria. These few men at some time or other during the course of their disease had positive Kahn tests, and one man had negative Kahns prior to treatment and positive Kahns during treatment.

THE FALSE POSITIVE SERODIAGNOSTIC TEST

In this laboratory, the tests which were found to be falsely positive became negative within a period of 4 weeks. The remaining men of the group that had positive tests at the time of entrance into the station, continued having strongly positive Kahns throughout the 10- to 12-week period of training. We are not prepared to state that more than 10 of the men having persistent positives had acquired syphilis or that more than 3 of them had congenital syphilis. The remainder of the group would require more intensive study than the time or the facilities allowed. They were informed of the findings prior to their discharge from the service, given no treatment, and told to report to their family physician at home for further investigation.

False positive tests may be classified in the following three types: (a) Those due to laboratory errors. These are a possibility in any laboratory and are the most likely cause when the recheck test a day or so later is found to be negative. Approximately 10 percent of our false positives were negative on recheck. This matter has been further investigated to rule out the possibilities of blood samples being transposed. The seriousness of the proper handling of the samples must be realized since transposition of blood samples may result in an actual

¹ Barnard, R. D.: *Ill. Med. J.* 77: 78-79, January 1940.

luetic being allowed to escape and a non-luetic being reported as positive. (b) Biologic false positives occur as a result of the unexplained presence of the antibody in the normal blood. (c) Those associated with some pathological condition other than syphilis. The following conditions have been set forth as possible causes of false positive diagnostic tests: Yaws, malaria, leprosy, relapsing fever, pellagra, scarlatina, bacterial endocarditis, infectious mononucleosis, Vincent's, malignancies (especially carcinoma of the tongue) rat bite fever, glanders, leishmaniasis, Weil's disease, lymphopathia venereum, chancroid, typhus, tuberculosis, upper respiratory infections, trypanosomiasis, vaccinia, spotted fever, acute lupus erythematosus, furunculosis, anemias, serum injections, epilepsy, obesity, menstruation, and chemical causes such as acetic acid poisoning, ether anesthesia, and during treatment with sulfanilamide and its derivatives.

It is believed that false positives occur in about 76 percent of those with leprosy, 21 percent of those having malaria, 7 percent of those having tuberculosis, 9 percent of those having malignant disease, 9 percent of those having miscellaneous fevers, and 4 percent in those with jaundice.² There are numerous reports in the literature that may indicate these figures are too low for malaria, too high for tuberculosis, and too high for those having general fevers.

THE KAHN TEST

Mitchell³ has reviewed the values of the diagnostic tests and points out that the Kahn test is of exceptional value because of its low incidence of false positives. He mentions the results during the League of Nations trials in Copenhagen in 1928 when 957 Kahn tests gave no false positives, and noted that similar results were found at Montevideo in 1930, and by the United States Public Health Service in 1935. Stokes⁴ states that the test used should have a specificity of at least 99 percent and a sensitivity of at least 65 percent, and that the latter in any laboratory should be approximately the same as in the originator's laboratory.

In these 20,000 Kahn tests, it has been found that the specificity ranges well above the required 99 percent, and, from coinciding results with Kahn's own laboratory on known positive bloods, it is believed that the Kahn test meets the requirements at the present time.

THE KAHN VERIFICATION TEST

Kahn⁵ considers the verification test as supplementary to the diagnostic test. The test is based on the belief that syphilitic serum gives

² *Urologic and Cutaneous Review*. 44: 504-600, September 1940.

³ Mitchell. *Colo. Med.* 34: 311-316, May 1940.

⁴ Stokes, J. H.: *J. A. M. A.* 112: 1132-1143, March 25, 1939.

⁵ Kahn, *Public Health Reviews*, Univ. of Michigan. 10: 53, June 1940.

a more marked reaction at 37° C., and that the biologic type of reaction is more pronounced at 1° C. The types of results are outlined as follows:

Positive Sera

Syphilitic type—more marked at 37° C.....	(common)
Biologic type—more marked at 1° C.....	(infrequent)
Indeterminate type.....	(infrequent)

Negative Sera

No precipitation at 1 or 37° C.....	(common)
Biologic type.....	(infrequent)
Syphilis, treated cases.....	(infrequent)
Inconclusive type.....	(uncommon)

Our experience with the test is insufficient to arrive at any conclusions. There were 10 samples of blood in our last group submitted to the University of Michigan at different times. Eight results corresponded to the physical and serological findings, while 1 was noted as syphilitic in type in a false positive, most likely caused by vaccinia, and another was reported as biologic in type in a man that was very strongly suspected of having acquired syphilis.

TITER AND PERSISTENCY OF TESTS

Moore⁶ has brought out that false positive tests are usually low in titer, while syphilitic bloods are usually high in titer. Barnes⁷ believes that if all tests are persistently positive, the possibility of a luetic infection is great. He adds that interpretation must not be made without corroborating evidence from the man's history as well as the clinical findings.

CONCLUSIONS

Observations on these 20,000 initial Kahn tests point to the warning that in the absence of definite history or physical findings, there is no justification for suspecting syphilis in those who have positive tests until 4 to 5 weeks of continuous testing has elapsed. Primary and accelerated cowpox reactions appear to be capable of producing false positive Kahn tests. The incidence of such false positive tests is low. No relationship has been demonstrated between antityphoid inoculation and the diagnostic Kahn test.

⁶ Moore, J. A. M. A. 115: 1602-1606, November 9, 1940.

⁷ Barnes, M. E., J. Iowa State Med. Soc. 30: 431-433, September 1940.

FALSE-POSITIVE WASSERMANN REACTION IN CEREBROSPINAL FLUID

CASE REPORT

By Lieutenant (Jr. Gr.) Russell L. Johnsrud, Medical Corps, United States Naval Reserve

Authentic false-positive spinal fluid Wassermann reactions may occur spontaneously in rare cases of intracranial tumor, encephalomalacia, trauma, cerebrospinal rhinorrhea, electric burn, or intrathecal neuritis; * * *.¹

It is the purpose of this paper to record another case in this group. For a complete discussion of this phenomenon, the reader is referred to the work of McLean and Munger.¹

CASE REPORT

A logger, aged 26, was first seen December 4, 1936, complaining of attacks of convulsions. He stated that in 1930 he was thrown about 20 feet striking his head on a log, was apparently unconscious for a few minutes, and then after recovering was able to return to work. He had a headache for the next 3 weeks, then one night while asleep he had a convulsion followed by coma from which he could not be aroused for an hour. One month later he had a second convulsion, and then subsequent ones at increasing intervals. The attacks were usually preceded by an emotional upset, as anger, and accompanied by headache until the actual convulsion took place which was usually at night. Observers stated that the convulsions were always generalized (not the focal or Jacksonian type), and followed by coma. The tongue was usually bitten, but there was no incontinence.

During childhood, the patient had had measles, mumps, smallpox, influenza, and otitis media with subsequent impaired hearing on the right side. Physical examination was essentially negative except for markedly impaired hearing on the right. Hematology and urinalysis findings were within normal limits. X-ray examination of the skull was negative.

The patient was placed on phenobarbital with doses gradually increasing to 0.1 grams daily; however, he continued to have more convulsions which now began in the right hand and occurred when he was awake. On August 4, 1938, physical examination was still essentially negative except for the finding of slight hypohesia of the right arm. Blood Kolmer and Kahn were negative. The spinal fluid was clear with globulin negative, cell count 0, protein 43.4 mg. per 100 cc. The spinal fluid Kolmer was four plus. The 24-hour Lange was 0001210000. (Because of this unexpected finding, a careful inquiry was made at the laboratory, but did not reveal any evidence of technical error. Controls and other tests made at the same time with the same method and reagents were apparently correct. This particular laboratory has always been very reliable.) September 13, 1938, spinal puncture was repeated with a specimen of fluid sent to the same laboratory, and another specimen to a second laboratory for checking. The Kolmer and Lange tests were negative in both reports.

On September 23, 1938, an encephalogram was made with the following findings noted in the radiologist's report. "There is a large area of atrophy that extends over the entire right lateral hemisphere to the posterior parietal region. The cerebral tissue is displaced away from the skull about 2 cm. There is also a large amount of atrophy present on the left side, although there is not so much displacement of the cortical tissue from the bone. The atrophy on the right side extends downward between the hemispheres. There is considerable air in this portion. The lateral ventricles are not enlarged, the right and third ventricles

¹ McLean, A. J. and Munger, I. C., Jr.: False-positive Wassermanns in cerebrospinal fluid. *Western J. Surg., Obs., and Gyn.*, 46: 455-462, September 1938.

are displaced to the right side by shrinkage from the atrophy. The aqueduct and fourth ventricles are normal. The 6-hour film shows a large amount of superfluous air over both lateral ventricles. The displacement is still present. There is still considerable air in the lateral ventricles."

The patient was examined on August 8, 1939, at which time the findings were essentially as before. He stated that he had been free of convulsions and felt much improved generally. During this time he had continued to take phenobarbital 0.1 grams daily.

SUMMARY

A case of post-traumatic cerebral atrophy is recorded in which a false-positive cerebrospinal fluid Kolmer reaction was obtained. This reaction should further be classified as a biological false-positive in contradistinction to a technical false-positive.

SULFANILYLGUANIDINE

TREATMENT OF ACUTE BACILLARY DYSENTERY¹

By Lieutenant Commander George M. Lyon, Medical Corps, United States Naval Reserve

INTRODUCTION

In a recent publication Marshall and his associates² have described an experimental study on sulfanilylguanidine. A remarkable feature of this chemical is that, although fairly soluble in water, it may be given in such dosages that saturation of the intestinal contents occurs without the blood concentration exceeding a relatively low level (1 to 4 mg percent). In a more recent publication³ they report encouraging results in its clinical use in the treatment of acute bacillary dysentery in a small series of infants and young children. The present communication describes an attempt to further test its efficacy by comparing a group of 23 patients with acute bacillary dysentery who were treated with sulfanilylguanidine with another group of 23 patients with acute bacillary dysentery who did not receive the drug.

BACKGROUND

In the tri-State area of West Virginia, Ohio, and Kentucky and in the adjoining mountainous mining regions, bacillary dysentery is endemic.⁴ For years it has been recognized as an important cause

¹ Presented for publication, January 14, 1941. This investigation was undertaken at the suggestion of Commander C. S. Stephenson (MC), U. S. N., Division of Preventive Medicine, Bureau of Medicine and Surgery, United States Navy. For the sulfanilylguanidine, the author is indebted to Dr. E. K. Marshall, Jr., Department of Pharmacology and Experimental Therapeutics, Johns Hopkins School of Medicine, Baltimore, and to the Calco Chemical Company, Bound Brook, N. J. Dr. Marshall also kindly provided the anhydrous monohydrate for use in preparation of the standard employed in the blood level determinations.

² Marshall, E. K. Jr., Bratton, A. C., White, H. J. and Litchfield, J. T. Jr.: Sulfanilylguanidine; chemotherapeutic agent for intestinal infections. *Bull. Johns Hopkins Hosp.* 67: 163-188, Sept. 1940.

³ Marshall, Bratton, Edwards and Walker: Sulfanilylguanidine in treatment of acute bacillary dysentery in children. *Bull. Johns Hopkins Hosp.* 68: 94-111, 1941.

⁴ Lyon, G. M.: Infant mortality studies in West Virginia. *W. Va. Med. J.* 30: 289, July 1934.

of morbidity. Fully 80 percent of all the cases of "bloody flux" occurring in these areas are due to the Flexner type of dysentery bacillus.⁵

Such a region provided opportunity for the study of this new chemical in the late summer and fall of 1940.

Through the cooperation of 24 physicians within this area it was possible to obtain access to a sufficient number of patients with acute bacillary dysentery to make the study possible. This included the majority of the patients with acute bacillary dysentery to be admitted to 8 of the larger hospitals of this area which extended for more than 75 miles to the north and west of Huntington, W. Va., and for more than 125 miles to the east and south of the same point.

PLAN AND CONDUCT OF STUDY

All patients were studied in accordance with a prearranged plan. Although it was not possible to hospitalize them all, nor even to have them all in the same locality, it was possible to obtain reliable observations on all patients included in the study.

As has been emphasized in a previous paper on chemotherapy in acute bacillary dysentery,⁶ it is important that the cases included in the study be of a relatively similar clinical character and severity. Acute bacillary dysentery may be a very mild disease. Because it occurs so frequently in this mild form in the United States, the actual cause of the illness is apt to be unrecognized.⁷ The majority of cases of acute bacillary dysentery occurring in the region in which this study was conducted are of the mild type. To include such cases in any study attempting to evaluate the efficacy of such a chemotherapeutic agent would seriously endanger the validity of the conclusions to be drawn, even when the presence of dysentery bacilli in the stools may have been proven by culture. In planning the study consideration was given to this important matter and relatively rigid criteria were established as prerequisites for admission to the group to be studied.

It was required that each patient selected present a reasonable certainty of having acute bacillary dysentery as shown by a history of acute onset with fever and diarrheal stools containing blood or pus, and that the rectal temperature on day of admission be not less than 102° and that the onset of diarrhea must not have been more than 7 days prior to admission. Although impossible to attempt stool cultures in many instances, diagnosis was confirmed by positive stool

⁵ Lyon, G. M.: Bacillary dysentery as observed among normal children in Huntington, W. Va. *Am. Dis. J. Child.* 49: 367, February 1935.

⁶ Lyon, G. M.: Chemotherapy in acute bacillary dysentery. Pt. I, Clinical types of acute bacillary dysentery. *W. Va. Med. J.* 37: 25, January 1941.

⁷ Holt and McIntosh: *Diseases of Infancy and Childhood*. Appleton-Century Co. New York, pp. 1099-1107, 1940.

culture in somewhat more than one-third of both groups. In the majority of the others, diagnosis was rendered somewhat more certain by finding the characteristic leucocytic exudation in the flecks of pus obtained from the stools when examined microscopically.

Patients were selected alternately for treatment with sulfanilylguanidine or for observation in the control group. Table 1 indicates distribution of patients by age group.

TABLE 1.—*Distribution of patients as to age groups*

Age in years.....	Under 2	2 to 6	11	13	33	75
Patients receiving drug.....	15	6	1	---	---	1
Patients not receiving drug.....	13	8	---	1	1	---

In the patients receiving the drug, urine examinations were made repeatedly in most instances in an attempt to recognize manifestations of toxicity. In many, rechecks of the urine were made as long as 1 month after treatment.

Temperatures were taken rectally in all but 3 instances. Temperatures recorded for day of admission represent maximum temperature observed on that day. Patients were considered to be without fever when the temperature remained consistently below 38° C. (100.4).

The stools were subjected to particularly close observation because of the importance they have in general, in indicating the trend of the clinical course of the disease. Particular note was made as to frequency and to character. When the stools were less than 5 per 24 hours, and were pasty or formed and contained no gross blood or pus the diarrhea was considered to be checked.

Observations as to the temperature curve, and as to changes in the frequency and character of the stools, and changes in the general clinical trend of the patients, were recorded in accordance with a prearranged plan, and in such a manner that uniform and systematic recordings were made. In a similar manner remarkable clinical behavior occurring in either of the two groups was noted.

The general treatment given the patients of both groups consisted of the usual supportive general measures with insistence on the maintenance of a good fluid balance and, when indicated, serum transfusions, whole blood transfusions, intravenous dextrose, etc. Laxatives were not permitted. Opiates and phenobarbital, singly or combined, were given for relief of tenesmus, pain and restlessness. Breast-fed infants were permitted to continue on breast milk. Whole cultured lactic-acid milk was given to the majority of the patients.

In general very bland, high protein, low fat diets were employed while much of the carbohydrate was given in the form of glucose orally. In the more severely ill patients of both groups, particular attention was given to the administration of oleum percomorphum, ascorbic acid, thiamin chloride, and nicotinic acid.

When sulfanilylguanidine was administered it was given in such manner that the patient received 0.1 gm. per kilogram of body weight as the initial dose and 0.05 gm. per kg. every 4 hours as a maintenance dose. When the stools became less than 5 per 24 hours, the frequency of administration was reduced so that the patient then received 0.05 gm. per kilogram of body weight every 8 hours for 48 to 72 hours thereafter. The drug was given in a suspension of milk or water, in some instances by oral gavage. In a few instances where vomiting occurred shortly after the drug was administered, half the amount previously administered was given at once in order to make up for the amount lost by vomiting. Fortunately, this was not necessary very frequently. In some instances beyond control, administration of the drug was discontinued before the time planned. Except perhaps in two instances (No. 17, No. 27) this appeared to be without effect on the general outcome of events. In a few instances, chemotherapy was continued beyond the time planned because, particularly in the earlier stages of the study, it was feared relapses might occur, so rapid and so difficult to comprehend had been the termination of the diarrheal state.

Because so little was known as to whether sulfanilylguanidine might produce toxic effects when used in this manner, a special effort was made to detect any such occurrences.

The patients included in both groups presented the clinical picture encountered with significant frequency by the physicians in the area in which the study was conducted. All of the physicians assisting in the clinical study, had previously had extensive experience with such cases and were peculiarly well fitted to supervise the general medical care of these patients, and to make reliable observations thereon. In addition they were peculiarly well qualified to express significantly trustworthy opinions as to the efficacy of the drug in this disease.

Cytologic examination of the blood was made at one time or another in roughly one half of both groups. In some of the patients receiving the drug such examinations were made repeatedly.

In several of the patients it was possible to study the concentration of the drug in the blood. This was done in accordance with the method advised by Marshall.¹ The standard employed was anhydrous sulfanilylguanidine. In infants and young children 0.04 cc. of blood

was precipitated in a 1: 200 dilution and in the older children and adults 0.5 cc. of blood, in a dilution of 1: 50. While blood concentrations are expressed in terms of the anhydrous compound, dosages are expressed throughout in terms of the monohydrate of sulfanilylguanidine. The concentration of sulfanilylguanidine in the blood varied generally between 1.5 and 3.6 mg. percent; the highest observed was 5.2 mg. percent in case 22.

SOME OF THE PROBLEMS OF THE STUDY

In attempting to find an index of the clinical activity of the disease in question, no single one, such as presence of fever or frequency and character of the stools, is by itself completely satisfactory. The general clinical trend of the patient is quite as important and this can hardly be reduced to graphic representation as can the other indexes.

In actual practice as the course of the disease progresses the general clinical trend tends to follow changes in frequency and character of the stools much more closely than the temperature curve. The duration of the fever and the duration of the diarrhea is presented in a graphic manner. The general clinical trend is described as clearly as possible in the text.

During the first few days of illness, fever, vomiting and bloody diarrhea are usually observed. Not infrequently, however, the first loose stools may not be passed until 24 to 72 hours after the onset of fever. For the sake of comparison and because it can be more accurately placed, the onset of diarrhea has been taken as the onset of illness. An attempt has also been made to record the date of onset of fever as accurately as possible.

During the recession of clinical activity of the disease, defervescence usually precedes improvement in the general clinical trend by an appreciable period of time. As ordinarily observed the patient continues to be clinically actively ill, sometimes severely and uncomfortably so, for some time after the temperature has returned to an essentially normal level. Since the stools may continue to be loose and to contain blood and pus for often as much as 2, 3, or more weeks after defervescence, the temperature curve cannot be taken as the sole indication of illness. Fortunately, the character and frequency of the stools tend rather closely to follow the general clinical trend in this stage of the disease and is a valuable aid to the clinician in the management of the patient.

GENERAL OBSERVATIONS AND CONSIDERATION OF DATA PRESENTED

The difference between the persistence of the febrile state and the diarrheal state in the patients receiving sulfanilylguanidine and those not receiving it is presented graphically (figs. 34 and 35); also the duration of treatment is so indicated. It is impossible to adequately

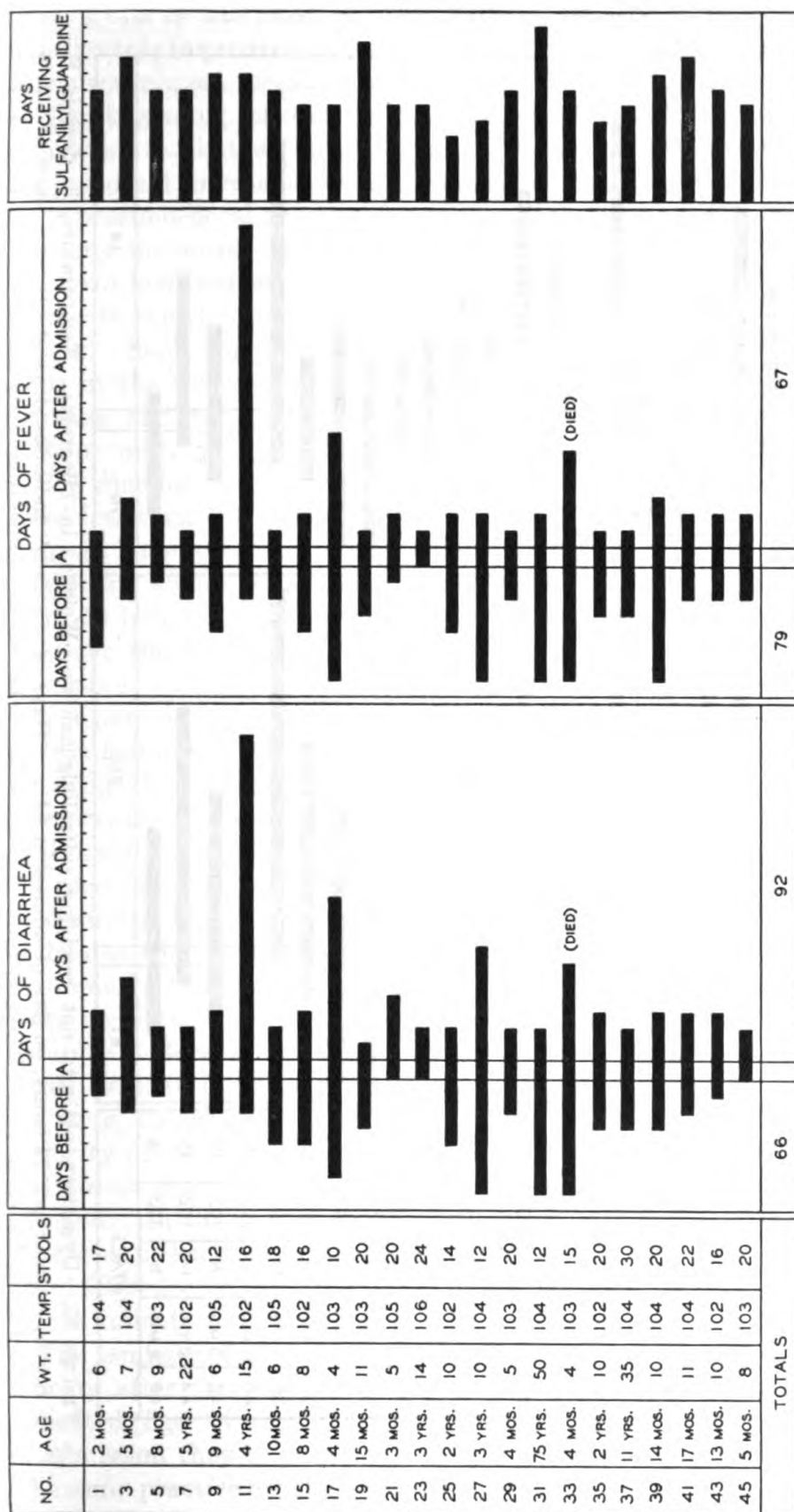


FIGURE 34.—Dysentery patients receiving sulfanilylguanidine—Weight, in kilograms. Temperature, in degrees F. Stools, number per 24 hours; on day admission (A). “Days of diarrhea” indicates days stools were loose and contained gross blood or pus. “Days of fever” indicates days when rectal temperature exceeded 38° C. (100.4 F.) at some time during the day. Treatment with sulfanilylguanidine instituted on day of admission (A).

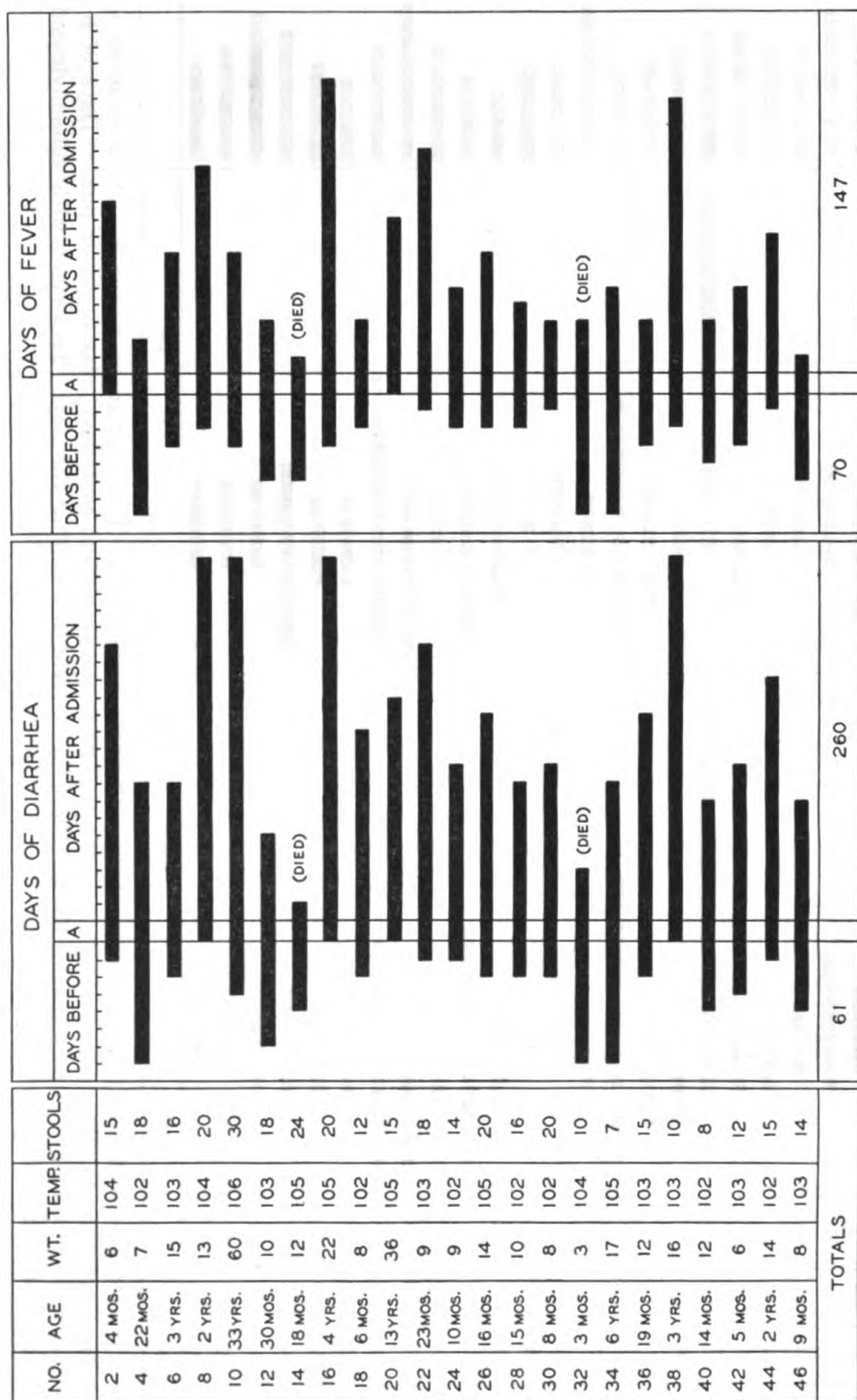


FIGURE 35.—Dysentery patients not receiving sulfanilylguanidine—Weight, in kilograms. Temperature, in degrees F. Stools, number per 24 hours; on day of admission (A). "Days of diarrhea" indicates days stools were loose and contained gross blood or pus. "Days of fever" indicates days when rectal temperature exceeded 38° C. (100.4 F.) at some time during the day.

present one of the most obvious features, namely the early, almost unbelievable improvement in the clinical picture and the clinical trend which was almost regularly observed to follow within 24 to 48 hours of the beginning of the chemotherapy. This effect can best be compared to that at times encountered in the treatment of some streptococcal infections with sulfanilamide and in the treatment of some pneumococcal pneumonias with sulfapyradine or sulfathiazole. In many instances the effect observed was as if an effective specific antitoxin had been administered, so striking was the rapidity and the degree to which the toxic manifestations of the disease would disappear. Such changes have never before been experienced by any of us in the treatment of acute bacillary dysentery. Generally the diarrhea and the indigestion of these patients will drag out over 2, 3, or more weeks under any form of treatment at our disposal.

The diarrhea was considered to be checked when the stools numbered less than 5 in 24 hours and were pasty or formed and contained no gross blood or pus. In the patients receiving the drug, the transition from abnormal to normal stools was generally rapid, within 48 to 72 hours after beginning chemotherapy, while in the group not receiving the drug after a period of active diarrhea of a week or more this same transition from abnormal to normal stools was prolonged over a period of 3 to 7 days or more, and the end point was never as clear cut as in the other group. In those patients who did not receive the drug, the majority, even after this transition period, continued to pass occasional stools that were softer than desirable and contained at times a little blood or pus. Frequently in these patients who did not receive the drug, during the period of transition the first stool in the morning would be soft, formed, and fairly normal and then later in the day there would be one or more stools with a little blood or pus, particularly the latter. Transitions of this kind were never seen in those receiving the drug, for there the change was made relatively abruptly. Because of this tendency to have occasional abnormal stools even during the period of convalescence in those not receiving the drug, the graphic description (fig. 35) errs in that probably the majority of the patients not receiving the drug had occasional abnormal stools after the time the chart indicates that the diarrhea was checked. In the case of those receiving the drug, in the majority of instances, no such abnormal stools were passed after the time indicated. The difference in this respect is therefore more marked than the charts indicate.

The tendency for the temperature to return to a normal level more quickly after treatment with sulfanilylguanidine is also graphically indicated (figs. 34 and 35). Where temperatures are shown for day of admission they refer to maximum temperature recorded that day. The same practice obtains in the case reports.

In a great many of the patients who received the drug and in whom an appreciable leucocytosis occurred, the leucocyte count tended to return to a normal level within 24 to 48 hours after beginning treatment with the drug. Apprehensive lest this might be an evidence of an aggressive action of the drug on the leucocytes, particular attention was paid throughout to the blood changes. Subsequent studies indicated that such was hardly the case and that the drop in the leucocyte count closely paralleled the improved clinical condition, particularly the disappearance of the toxic manifestations. In one instance (No. 22) it was for a time feared that an agranulocytosis might have been initiated by the use of the drug. More careful consideration of her case history and her subsequent improvement under chemotherapy tend to deny rather than to confirm this. In two instances, (No. 11, No. 22) as will be described under the resume of case reports, a hemolytic anemia may have been aggravated by the drug, and could quite as well have been caused primarily by an associated pyogenic infection.

There was a striking absence of toxic symptoms, certainly those of any significance, in the patients receiving the drug.

Of the 23 patients so treated, only 3 evidenced some tendency to nausea and vomiting, which it was believed might be due to the drug (No. 11, No. 25, No. 33). Such symptoms were quite as common in the control group as in those treated. In one instance, there may have been some disturbance of the sensorium and some agitation (No. 25), and in one instance, there may have been some conjunctival injection (No. 37). In two instances (No. 11, No. 13) it was felt there may have been some discoloration of the skin such as is seen so frequently in treatments with sulfanilamide, although this was not remarkable. In 8 of the infants so treated, the stools were black and much like meconium (No. 29, etc.), for the first 2 to 3 days they received the drug. The meaning of this, if any, is not known.

In general it may be said that toxic effects, immediate or late, were much less severe and were seen with much less frequency than would have been expected with either sulfanilamide, sulfapyradine or sulfathiazole. No tendency to acidosis or renal irritation was observed. The drug appears to be more effective if given early in the disease, failures being few when chemotherapy was initiated within the first 3 to 5 days of the onset of diarrhea. This effect is illustrated in table 2.

TABLE 2.—*Influence of early administration on effectiveness of drug*

Days of diarrhea before patient received drug.....	0	1	2	3	4	5	6	7
Successful.....	3	3	4	4	3	0	0	1
No influence.....	0	0	2	0	0	0	1	2

CASE REPORTS

PATIENTS WHO RECEIVED SULFANILYLGUANIDINE

Of those who received sulfanilylguanidine, several merit description, and the first may be taken as typical of those of the group who manifested remarkable benefit from the drug.

Patients Benefited

No. 35.—C. H., 2 yrs, 10 kg., was admitted to the service of Dr. T. Folsom November 11 with a complaint of vomiting, fever and bloody diarrhea for 3 days. His temperature was 102° and he had 20 or more loose stools containing blood and pus during the day preceding. He was first given sulfanilylguanidine on the day of admission. There was a remarkable improvement in his general clinical condition and his temperature returned to normal within 24 hours. His stools were normal after 72 hours. The stools were free from blood after the first 24 hours. There was a rapid decrease in the number of stools per day and a great improvement in the character of the stools within 24 hours after chemotherapy was begun. Dr. Folsom said, "He was essentially well in 3 days. The result was dramatic. His convalescence was very rapid." He received the drug for only 5 days. The stools were not cultured. Special blood studies were not made.

No. 1.—A. R., 2½ mos., 6 kg., breast fed, was admitted to the service of Dr. Lyon, Memorial Hospital on September 21. Because of distention and vomiting, the infant was suspected of having intussusception or intestinal obstruction, and for this reason was referred to the hospital. Curiously enough, illness had been initiated with a convulsion. The temperature was 103°–105° during the first 5 days in the hospital. At the end of this time (September 25), loose bloody stools were passed. The following day 17 bloody stools were passed and the maximum temperature was 104°. Sulfanilylguanidine suspended in 10% dextrose solution was then and thereafter given by gavage. Although the infant had been unable to nurse vigorously it was possible to give the milk, expressed from the mother, by gavage. The infant was critically ill. Dehydration was combatted mainly by frequent oral gavage of 10% dextrose in physiologic saline solution. This was well retained. Within 24 hours after the drug was first given, the temperature fell to a nearly normal level, and there was a similar dramatic improvement in the general clinical condition. After 72 hours of chemotherapy, the stools were normal breast milk stools. The Flexner bacillus was recovered from the stools passed on the day the drug was first given. September 25, WBC 17,000. September 28, WCB 11,000. October 2, WBC 9,000. The drug was given for a period of 7 days. This rapid recovery is remarkable because of the age of the patient, and the severity of the infection.

No. 29.—M. Z., 4 mos., 5 kg., was admitted to the service of Dr. W. Parsons, Huntington Baby Clinic on October 25. There had been a bloody diarrhea and fever for 2 days. On the day of admission, the baby had a temperature of 103°, was quite ill, and had passed 20 or more bloody stools the 24 hours previous. Parents were intelligent and cooperative. Miss Sprouse, the visiting nurse from the clinic, closely supervised the treatments. The drug was given at times by oral gavage and at times just suspended in the whole cultured lactic acid milk and given from the bottle. Within 24 to 48 hours, a most striking improvement occurred. The temperature had returned to normal. The stools were normal and pasty after the first 48 hours of chemotherapy. The drug was administered for 7 days, in accordance with the general plan. The visiting nurse who has had

more than 12 years experience with such patients said, "I have never seen anything like it. She was well by the third day." Two weeks after beginning chemotherapy her blood examination was normal. She was watched particularly carefully because on the first two days she received the drug, her stools were very similar to meconium. Reference as to this having occurred in several of the infants so treated, has been made.

No. 25.—D. B., 3 yrs., 14 kg., was brought to the office of Dr. Lyon on October 10, having just had a convulsion. Prior to this he had been quite well. His temperature was 106°, and he passed his first loose stool, which contained blood, while in the office. During the next 24 hours he had on an average of one or more such stools every hour. They were mostly blood and contained little fecal matter and little pus. He continued to be quite ill and to be quite prostrated. On the second day of chemotherapy, his temperature had returned to normal and he had his last diarrheal stools within 48 hours of his first dose of sulfanilylguanidine. His general clinical improvement was one of the most dramatic of the entire series. On the sixth day of treatment, which was the sixth day after onset of his diarrhea, he was a well boy, on a normal diet and having normal physical activity. Day of onset: (October 10) RBC 4,100,000, WBC 7,800. Two weeks later his condition continued to be entirely normal, and a recheck of his blood for purpose of our study revealed RBC 3,900,000, Hb. 69% (Sahli), and WBC count 14,000. The differential smear was not remarkable. His stools were never cultured. That he had little or no pus in his bloody stools is a noteworthy fact. The clinical course was rapidly terminated.

No. 31.—O. W., 75 yrs., 50 kg., was admitted to the medical service of Dr. C. E. Richards, Holzer Hospital, Gallipolis, Ohio, on November 1. He had had a severe bloody diarrhea with vomiting, chills and fever for 10 to 12 days previous to admission. The Flexner bacillus was recovered from his stools. He was severely dehydrated and gravely ill. Besides his chemotherapy, he was given intravenous glucose and saline solution. His abdominal cramps were severe. He had 12 stools with blood, pus and mucus the first 24 hours of admission, and his maximum temperature that day was 104°. Proctoscopic examination revealed the characteristic findings. He was given a typhoid diet. Through an error, after an initial dose of 6.0 gm. of sulfanilylguanidine, he received for the next 2 days only 3 grains every 4 hours instead of 3.0 gm. which he was supposed to receive, and which he did receive every 4 hours after this time. That he received a remarkable benefit from his initial dose, and perhaps even some from the small amount he received thereafter for 2 days, is evidenced by the fact that his stools dropped from 12 per day to 4 per day within 24 hours; although they did contain a little blood and pus for a second 24 hours period, the improvement was remarkably rapid and they were essentially normal the following day. His temperature was normal after 48 hours of chemotherapy. He was remarkably less toxic after only 24 hours of drug treatment. His general clinical picture showed an equally striking improvement in the first 48 hours, and he became very anxious for food. He received the drug for 11 days. The blood concentration varied from 1.5 mg. percent to 2.2 mg. percent, the latter on the fourth day of treatment. On discharge, his blood examination revealed RBC 4,000,000, Hb. 76 percent, WBC 6,600 (neutrophils 52 percent, lymphocytes 32 percent, and monocytes 16 percent). He showed no urinary abnormalities. His blood nonprotein nitrogen was 35, blood sugar 100, and chlorides 480. His Widal and Wassermann were both negative. He had a rapid uneventful recovery and has had no recurrence of his trouble since discharge. This case is of importance because it demonstrated that even at relatively late stages in the disease, sulfanilylguanidine may, at times, effect quite as dramatic results as when used earlier. The age of

the patient as well as the severity of his infection likewise adds to the interest of the case.

Patients Not Benefited

No. 11.—H. E., 4 yrs., 15 kg., was admitted to the service of Dr. F. A. Scott and Dr. T. G. Folsom, St. Mary's Hospital, on October 3, for an operation for an old osteomyelitis of the tibia. Four months previously this patient had been admitted to the hospital to receive treatment for this same disability and during that stay had received at one time or another both sulfanilamide and sulfathiazole. On the first day of his second admission, it was found that he had a bloody diarrhea. Treatment was begun at once so that he received sulfanilylguanidine 2 days after the onset of his bloody diarrhea. He was not very ill. In fact, much of his illness was probably due to his osteomyelitis, rather than to his bacillary dysentery. Besides this, he had a significant ascariasis which was not given therapeutic attention at this time. He received the drug over a period of 8 days (October 3–10) and was apparently not benefited by it, as his fever and purulent, bloody diarrhea continued.

Note blood findings thereafter:

October 3	-----	RBC 5,140,000 Hb 78 percent WBC 13,700
11	-----	RBC 4,240,000 Hb 70 percent
16	-----	RBC 2,780,000 Hb 37 percent WBC 53,000
18	-----	RBC 3,180,000 Hb 62 percent WBC 31,000
24	-----	RBC 3,260,000 Hb 66 percent WBC 12,400
30	-----	RBC 3,210,000 Hb 66 percent WBC 18,800

While the diarrhea persisted, it was not as severe as is usually encountered in a case of such apparent gravity. Several blood transfusions (October 11–16) improved his blood picture somewhat and the subsequent, but more or less accidental, administration of sulfathiazole 3 weeks later was followed by an immediate cessation of his bloody and purulent diarrheal stools. This was not accompanied by any depression of either erythrocytes, hemoglobin, or leucocytes. He continues to have a clinically unsatisfactory course probably due to his osteomyelitis. Except for 3 weeks recently, he has been febrile (100.4°–102°) much of the time the past 3 months, that is, since the onset of the dysentery infection, although this has long since ceased to be a part of his clinical picture. At various times throughout his clinical course he has had red blood cells, casts, and albumin in his urine. This was not apparently related to his chemotherapy. It is likely that he should not have been admitted to this group for study, because of his complicating osteomyelitic infection. It was the impression of Dr. Folsom that the hemolytic anemia was at least aggravated by, if indeed, not induced by his chemotherapy. There are too many disconcerting factors to make this seem a certainty. No agranulocytosis occurred.

No. 17.—E. B., 4 mos., 4 kg., was admitted to the service of Dr. J. Basman, Charleston General Hospital, Charleston, W. Va., on October 8, with a history of fever and bloody diarrhea of 6 or 7 days duration. Sulfanilylguanidine was first given on the seventh day of diarrhea. Although the drug was given in the prescribed manner, the patient continued to have a temperature of 102°–103° for a week, and it was 10 days or so before the stools were greatly improved. Dr. Basman stated that he noted no change following the use of the drug. This was an unquestioned failure, but treatment was begun at the end of the first week, a fact which may have some significance.

No. 27.—L. A., 3 yrs., 10 kg., was admitted to the service of Dr. J. Basman, Charleston General Hospital, Charleston, W. Va., on October 21. There had

been fever and bloody diarrhea for 10 days. The drug was first administered on the tenth day of the diarrhea, at just about the time one might expect a natural recession of the disease to occur. The drug was administered for a period of 4 days only, during which time the natural improvement apparently took place. Dr. Basman said, "I do not believe it was a fair trial, but I saw no improvement in the bowel movement after the drug was given." It was unfortunate that this case had to be included, but it was necessary to do so or upset the sequence of the series. It was planned to accept only patients who had had their diarrhea for less than 1 week, and this patient had diarrhea for 10 days prior thereto.

No. 33.—H. R., 4 mos., 4 kg., was admitted to the service of Dr. Lyon, St. Mary's Hospital, on November 4, with a bloody and purulent diarrhea of 7 days duration. The patient was extremely emaciated and was said to have done poorly from birth. He was admitted in a critical condition. Chemotherapy was begun promptly on admission. He received no benefit from the drug so far as the purulent diarrhea was concerned, and developed a pneumococcic meningitis from which he died on the sixth day after admission. Again, this was not a desirable type of patient to have included in such a series, and while admittedly the drug was not observed to be effective, bacillary dysentery occurring in a poorly nourished infant of 4 mos. was attended by a severe and fatal complication outside the gastrointestinal tract. Stool cultures were not obtained. The microscopic examination of the purulent material from the stool was highly significant and rendered a diagnosis of bacillary dysentery reasonably certain.

No. 3.—E. S., 5½ mos., 7 kg., was admitted to the service of Dr. Lyon, Memorial Hospital, on September 26, with a history of fever and bloody diarrhea, which had appeared 2 days before. Following the admission and the administration of the drug, the fever persisted for 3 days and the diarrhea for 5 days. As the parents insisted on removing the baby to the home on the same day it was admitted to the hospital, and since they were not entirely dependable as to care and medications, the thoroughness of the administration of the drug is not known. Two doses a day were, however, regularly given by a visiting nurse, for a period of 9 days. In this instance, the drug had no dramatic effect, and indeed may have had no effect. Because of this uncertainty and because it was one of the 5 of the 23 patients treated with the drug who continued to have a diarrhea for more than 3 days after beginning treatment with the drug, the result of this treatment is entered as a failure. On the other hand, one cannot be certain that it was entirely without effect, for the stools were normal on the sixth day after treatment with the drug was begun.

Patients Manifesting Important Toxic Symptoms of Questionable Significance

HEMOLYTIC ANEMIA.—The description of Dr. Folsom's patient, H. E. (No. 11), has been given among those of the patients who failed to benefit from the drug.

AGRANULOCEYTOSIS.—No. 22.—L. H., 23 mos., 9 kg., was a patient of Dr. Richards of the Holzer Hospital, Gallipolis, Ohio, and was admitted on October 10, as a member of the control group. She was permitted to have the drug after 14 days of bloody diarrhea and fever. There was a lesion of the left external nasi which involved the nasal septum and the lip, and it was at first feared this might be a beginning noma. The stools were positive for the Flexner bacillus. October 26, the day following the beginning of chemotherapy, RBC 3,440,000, Hb. 58 percent, WBC 3,800 (neutrophils 28 percent, lymphocytes 56 percent, monocytes 12 percent, and myelocytes 4 percent). Two days later the blood sulfanilylguanidine was 5.2 mg. percent. Blood examination at that time revealed, RBC 2,840,000, Hb. 52 percent, WBC 4,200 (neutrophils 20 percent, lymphocytes 80 percent). After 7 days of treatment, the drug was discontinued. At this time examination of the blood revealed RBC 2,720,000, Hb. 47 percent, WBC 10,800,

(neutrophils 36 percent, lymphocytes 58 percent, and monocytes 6 percent). The chemotherapy was followed by a rapid improvement in her general condition. Within 48 hours she was much less toxic. The diarrheal stools had dropped from 12 to 5 and their character improved with equal rapidity. She made an uneventful recovery and has remained entirely well since. The lesion on the lip had disappeared completely by the time of discharge from the hospital. The rapidity with which the blood changes occurred suggest that this may have been a part of her original disease (dysentery or infection of lip). This appears more likely. One notes the drop in the blood from that of October 21, 4 days before the drug was given (RBC 4,400,000 Hb. 76 percent, WBC 43,000, PMN 75 percent, lymphocytes 22 percent, monocytes 3 percent), to that reported above for October 26, less than 24 hours after beginning chemotherapy. It is unlikely that the action of the drug was responsible for these changes in her blood. Whether the drug was an aggravating factor cannot be said.

PATIENTS NOT RECEIVING SULFANILYLGUANIDINE

Of the patients in the control series, who did not receive the drug, the clinical course of three should be described; the first, F. R. (No. 6), because the infection and the clinical course was the least severe and of shortest duration of any of the group, the second, J. K. (No. 16) because hers was one of the most severe of those to survive, and the third, J. W. (No. 44) because his clinical course typified that of the average severity and duration for the group. There were 2 deaths in this group (No. 14, No. 32).

No. 6.—F. R., 3 yrs., 15 kg., was admitted to the service of Dr. Parsons, St. Mary's Hospital, on September 15, with a history of having had fever for three days and bloody diarrhea for two days. On the day of admission, there were 16 bloody stools with little pus and the maximum temperature was 103°. The patient had fever for seven days after admission, and dysenteric stools with blood and pus, or pus, for at least eight days after admission. Her febrile course was 101°–103° during her period in the hospital. There was from the beginning a disinclination to take food or fluids, and during the second week after the onset, she had a slow but steady return to normal stools and a return to a more normal general condition. Convalescence was hardly completed by the end of the third week. The Flexner bacillus was recovered from the stools. Three patients (No. 12, No. 28, No. 30) presented cases of significantly more severity. The remainder of the control group were of a much greater severity and of longer duration.

No. 16.—J. K., 4 yrs., 22 kg., was admitted to the service of Dr. Lyon on October 7. She had fever for three days prior to admission but had become gradually worse until it reached 105° on this day and shortly thereafter she began passing loose bloody almost hemorrhagic stools. At the time she was very hyperesthetic and vomitted several times. Throughout the following week she was quite ill, at times delirious. The stools were more than 18 to 20 daily and contained no pus until the third or fourth day of diarrhea. Tenesmus was intense and required much opiates. There were occasional chills. Persistent, attentive nursing obviated the necessity of gavage or intravenous fluids. A splendid fluid intake was maintained. The diet was limited to cultured lactic acid milk, and cottage cheese during the active stage. She was not afebrile until 18 days after admission and the stools contained pus and mucus with occasional blood throughout the third week. Prolapsus ani was severe and persistent. At the end of the third week, while much better and the convalescence beginning, she was still very weak, had lost much weight, and a good month was required thereafter before she returned to a relatively good physical condition. Leucocytic forms were observed in the purulent flecks from the stools. The Flexner

bacillus was recovered from the stools. Blood counts October 4, WBC 12,000, October 7, WBC 20,000, October 10, 22,000, October 17, 18,000. Her case was one of the severe form.

No. 44.—A case of somewhat above average severity for the group, was that of J. W. 2 yrs., 14 kg., admitted to the service of Dr. W. B. Hunter on November 22. Onset was sudden with fever, vomiting, and diarrhea 24 hours before admission, at which time the temperature was 103.4°. There had been 13 bloody stools the 24 hours previous. The patient was quite actively ill during the first week. There continued to be fever, 101° to 103°, for 8 days after admission. The stools were loose and contained blood and pus, or pus, for 14 days after admission. There was the usual anorexia, tendency to nausea, loss of strength, and ennui. There were from time to time thereafter occasional stools which were not quite normal, and there was the usual intestinal sensitivity to too much food and to harsh foods. Convalescence was rather well advanced by the end of the third week.

No. 14.—D. W., 18 mos., 12 kg., was seen by Dr. Lyon in consultation at the patient's home in rural Ohio on October 4. There had been fever and bloody diarrhea for 5 days. The temperature was 105° and the stools were passed almost constantly. The dehydration was severe and the condition genuinely critical. Hospitalization was refused. The clinical picture grew steadily worse. Death occurred late the following day.

No. 32.—R. C., 3 mos., 3 kg., was first examined by Dr. Lyon on November 1. The complaint was fever, vomiting, and diarrhea in an infant which had never done well from birth. The temperature was 104°, the stools had been loose and with blood in them for 7 days. The infant was in a critical condition and in spite of the usual measures of dextrose solution, serum transfusion, etc., death occurred three days later.

SUMMARY

Twenty-three patients with severe acute bacillary dysentery were treated with sulfanilylguanidine, and a like number of patients with the same disease and with cases of a similar severity who did not receive the drug, were taken as controls. Alternate patients were treated.

In order that the cases be of as similar a clinical character and severity as possible, rather rigid criteria were established as a prerequisite for selection. The importance of this was emphasized.

Of the patients who did not receive sulfanilylguanidine, the characteristic clinical course with which we are so familiar was observed. Temperature of 102–105° F. for the first week with nausea, vomiting, severe bloody diarrhea, abdominal cramps, tenesmus, prolapsus ani, and in the second week a lower temperature, a continuation of the diarrhea which becomes more purulent than bloody, and a loss of strength and body weight. In the third week, a tendency to enter a convalescence of varying degrees of severity and tardiness, a clinically significant intestinal indigestion and continued weakness are the important features.

Of the patients who received sulfanilylguanidine, 5 may be said, with certain reservations, not to have been benefited, while 18 were

strikingly benefited. Many of these recoveries were most dramatic in character. Generally, after the institution of chemotherapy, a fall in the temperature and in the leucocyte count was observed within 24 to 48 hours, as well as a closely paralleling general clinical improvement, a marked reduction in the number of diarrheal stools, and within 48 to 72 hours a remarkable improvement in the character of the stools as to freedom from blood, pus, or mucus.

The drug appears to be most efficacious when administered during the first 3 to 4 days after onset of fever or diarrhea. Although the percentage of failures may be higher when the drug is not given until after the fifth day of illness, it has been observed to be effective at times even when given later in the disease.

Observations at hand lead to the assumption that sulfanilylguanidine is an effective agent for the treatment of acute bacillary dysentery, that it should be used with conservatism and that conclusions should be guardedly drawn. Toxic effects possibly due to the drug may have been observed in the blood of two patients, who concomitantly had clinically significant infections outside the intestinal tract. Generally speaking, the use of the drug was characterized by freedom from toxic effects in the dosages employed. The toxicity of the drug appears to be less than that of the related compounds sulfanilamide, sulfapyradine, and sulfathiazole.

CONCLUSIONS

1. Sulfanilylguanidine is effective in the treatment of acute bacillary dysentery, or "bloody flux."
2. It appears to be most effective if administered within the first 3 or 4 days of the diarrheal state.
3. Toxic manifestations, whether immediate or remote, appeared to be much less frequent and of much less significance than those encountered in the use of either sulfanilamide, sulfapyradine, or sulfathiazole.

NOTES AND COMMENTS

WILLIAM CLARENCE BRAISTED

By Commander Louis H. Roddis, Medical Corps, United States Navy

William Clarence Braisted, the nineteenth Chief of the Bureau of Medicine and Surgery, and the fifteenth in succession to hold the office of Surgeon General of the United States Navy, was born in Toledo, Ohio, on October 9, 1864, less than a year before the assassination of President Lincoln. Of equal or more interest is the fact that he was the first Surgeon General of the Navy to come from west of the Allegheny Mountains, all others before him having been from one of the original thirteen colonies. In 1883, when but 19 years of age he graduated from the University of Michigan and, in 1886, he received his degree of Doctor of Medicine from the College of Physicians and Surgeons, of Columbia University, standing first in his class. He served 2½ years as a house surgeon at Bellevue Hospital, and practiced for 2 years in Detroit, Mich., before entering the Navy as an assistant surgeon on September 26, 1890. His first commission was signed by President Cleveland. His first duty in the Navy was far from either ocean, for he was sent to the Army and Navy Hospital at Hot Springs, Ark. Doctor Braisted was asked to make a complete analysis of the waters used there both for baths and internal medication.

His first sea duty was on the celebrated dynamite gun ship, appropriately named the *Vesuvius*, the only ship of its kind ever operated in full commission in any navy. It carried three pneumatic guns extending nearly the whole length of the ship and designed to hurl huge charges of dynamite at an enemy. The vessel was unsuccessful and after many trials was finally given up. While serving on the *Vesuvius*, Doctor Braisted was decorated by the President of Venezuela for caring for the wounded after a battle at Puerto Cabello during a revolutionary outbreak. Altogether he had 9 years of sea duty on various vessels, including the *Vesuvius*, *Detroit*, *Topeka*, *Massachusetts*, *Connecticut*, *Utah*, and *Wyoming*. He was operating surgeon at New York Naval Hospital and twice instructor in surgery at the Naval Medical School, Washington, D. C. One of his most interesting experiences was during the Russo-Japanese War when he served as an observer with the Japanese. His report on the medical and surgical features of this war is a model of what such reports should be and is a most interesting as well as informative narrative.

From 1906 to 1912, he served with Surgeon General Rixey as an attending physician to the White House. From the latter date until 1914, he was fleet surgeon of the Atlantic Fleet. He was appointed Surgeon General on February 7, 1914, by President Wilson. He was thus Surgeon General during the trying times before, during and after the World War I, and faced the tremendous tasks thrown on the naval medical department when the expansion of the Navy from 55,000 officers and men, to over half a million took place, as well as demobilization to peace strength after the war. The problems of the war including the great influenza epidemic of 1918 had to be met by the Medical Corps under his leadership. He was one of the foremost workers in the Council of National Defense during the war. Dr. Franklin Martin said of him in his book in describing the work of the Council, "Doctor Braisted of the Navy, was always a joy. His Bureau of Medicine and Surgery ran along on carefully oiled wheels, and with the details of whose operation, this master steersman was in constant touch. His department was always several jumps ahead of any emergency that arose. His storehouses were full, due to the vision of himself and his associates."

Doctor Braisted was President of the Association of Military Surgeons of the United States from 1913-14 and was the only Surgeon General of the Navy to serve as President of the American Medical Association, 1919-20. He was one of the most ardent advocates of a National Board of Medical Examiners and the present National Board owes much to his labors for its establishment and operation.

Doctor Braisted retired on November 29, 1920, and was President of the Philadelphia College of Pharmacy and Science from 1920 to 1925. He received many honors, including academic distinctions from a number of universities and honorary Fellowship in the American College of Surgeons. Decorations included the Distinguished Service Medal of the United States, the Order of Bolivar from Venezuela and the Order of the Rising Sun of Japan.

Doctor Braisted died at his home, West Chester, Pennsylvania, on January 17, 1941, and interment with full military honors was in Arlington National Cemetery.

SUMMARY OF ACHIEVEMENTS OF HIS REGIME

1. Compilation of a Hospital Corps Drill Book.
2. Commissioning of the naval hospitals, San Diego, California, Charleston, and Parris Island, S. C.
3. Designing and building of the U. S. S. *Relief*, the only hospital ship designed and built as such.
4. Surgeon General of the Navy during the World War I.



REAR ADMIRAL WILLIAM CLARENCE BRAISTED, MEDICAL CORPS, U. S. NAVY.
Surgeon General 1914-20.

ARTICLES OF SPECIAL MERIT, 1940

The Surgeon General takes this opportunity to express to all contributors to this Bulletin his satisfaction with the excellence of their articles and his appreciation of their support of the Bureau's publication.

It has become an established practice for the Surgeon General to present letters of appreciation to authors who have contributed articles of outstanding merit to the Naval Medical Bulletin. For the calendar year 1940 the following officers have received letters of appreciation:

Commander W. W. Hall (MC) U. S. N. and Chief Pharmacist P. S. Gault, U. S. N. *Advances in Clinical Chemistry*. January 1940.

Lieutenant Frederick R. Lang (MC) U. S. N. *Meningococcus Antitoxin in Cerebrospinal Fever*. January 1940.

Captain L. W. Johnson (MC) U. S. N. *Surgical High-Lights of 1939*. January 1940.

Lieutenant (Jr. Gr.) Earle E. Metcalfe (MC) U. S. N. *A New Method for Determining Night Blindness*. April 1940.

Commander Walter Rehrauer (DC) U. S. N. and Lieutenant (Jr. Gr.) Karl Van Lear Berglund (DC) U. S. N. *Dental Abnormalities in Recruits*. April 1940.

Captain Reynolds Hayden (MC) U. S. N. and Commander Orville R. Goss (MC) U. S. N. *Visual Deficiency, Incidence at the U. S. Naval Academy and Measures Taken for its Reduction*. April 1940.

Captain W. Chambers (MC) U. S. N. and Commander C. F. Behrens (MC) U. S. N. *Roentgen Photography*. July 1940.

Lieutenant Clifford F. Storey (MC) U. S. N. *Tumors of the Vermiform Appendix*. July 1940.

Lieutenant Commander Gerald W. Smith (MC) U. S. N. *Diagnosis and Treatment of Head Injuries*. October 1940.

Commander P. P. Maher (MC) U. S. N. and Lieutenant A. H. Staderman (MC) U. S. N. *Bronchogenic Carcinoma*. October 1940.

Commander E. G. Brian (MC) U. S. N. and Lieutenant E. Ricen (MC) U. S. N. *Surgical Treatment of Pulmonary Tuberculosis*. October 1940.

CEREBROSPINAL FEVER¹

Simplified instructions on the management of outbreaks of cerebrospinal fever have recently been promulgated by the British War Office. These are based on experience gained during an epidemic of the disease occurring last winter. The following epidemiological measures have been abandoned: (a) Segregation of contacts (these are recorded, however); (b) routine post-nasal swabs for the detection of carriers; (c) mass gargling and insufflation.

The revised outline of instructions is as follows: (a) Lectures to the men on mode of transmission, and on the dangers of droplet infection; (b) widespread exercise in open air urged; (c) avoidance of hot, stuffy, and humid rooms, and overcrowding; (d) maximum ventilation while

¹ Special Article: *The Lancet*, vol. II, p. 608, November 9, 1940.

rooms are vacated; (e) head to foot sleeping, with bed centers at least 6 feet apart.

With reference to treatment, sulfapyridine is believed to be the chemotherapeutic agent of choice for the reason that it is considered more effective than either sulfanilamide or sulfathiazole. Lumbar punctures are performed only for diagnostic purposes or to relieve pressure symptoms. Routine treatment is similar to that generally accepted in the United States (8 to 10 gm. given by mouth per 24 hours during the first 3 days in dosages spaced at 4-hour intervals). On the fourth day, the dosage is reduced to 2 to 3 gm. per day at the same intervals. Ordinarily, this medication is discontinued at the end of the ninth day. In cases of persistent vomiting, it was found that relief could be procured by substituting the same dosage of sulfanilamide. Either drug is administered to unconscious patients by means of nasal or oral tube. Because of its excess alkalinity, intravenous or intramuscular injection of the sodium salt is used only in fulminating or unconscious cases.

NEW MEMBERS, AMERICAN COLLEGE OF SURGEONS

The following named naval medical officers were nominated by the Bureau of Medicine and Surgery for fellowship in the American College of Surgeons and the Bureau has been informed that they were elected at the 1940 convocation of the College.

Lieutenant Lewis T. Dorgan, (MC), U. S. Navy.

Lieutenant L. D. Ekvall, (MC), U. S. Navy.

Lieutenant Louis E. Gilje, (MC), U. S. Navy.

Lieutenant Garland A. Gray, (MC), U. S. Navy.

Lieutenant Paul Peterson, (MC), U. S. Navy.

NOTICE

The Association of Military Surgeons invites naval medical officers to submit papers for consideration by the program committee with a view to presentation at the 1941 annual meeting of the Association at Louisville, Kentucky, on October 29th, 30th, and 31st.

THE NEUROSES IN WAR¹

To all physicians, particularly to those who are directly concerned with the psychiatric problems of war, this volume comes as a welcome and timely compendium. Within its covers are outlined the experiences of a number of men who are well qualified to speak of various aspects of the emotional stresses to which human beings, both civilian and military, are subject in time of war. A perusal of this work gives one as comprehensive an idea of the problem as can be gained through the reading of any one volume on the subject.

Of particular interest are the observations bearing on the military

¹ Edited by Emanuel Miller. The Macmillan Co., New York, 1940, see Book Review section of this

success of different personality types. Many authors find that individuals with a tendency to anxiety, phobic and depressive reactions, as well as those who are excessively timorous, have a poor military prognosis, whereas those of obsessional make-up not only do well, but also actually distinguish themselves in military service and only subsequently break down on return to their former civilian status. Another finding not sufficiently stressed among the psychiatric profession at large is here given some prominence, namely, the fact that family factors and heredity play a relatively unimportant role in assaying the military value of an individual from a psychiatric point of view. It might not be amiss to emphasize that the past achievement of the individual and the manner in which he reached his goals are factors of infinitely greater diagnostic significance.

In discussing the symptomatology of war neuroses the authors mention a number of prodromal signs such as fatigue, increased indulgence in alcohol and tobacco, tendency to become unsociable or irritable, loss of interest, disinclination for effort, emotional crises, crying fits, states of terror, and character changes which, if properly understood and evaluated, would help to reduce the incidence of frank break-downs.

Regarding the prognosis of functional disturbances developing under service conditions, the authors state that those whose history showed break-downs before enlistment or who had pronounced character difficulties, and showed a preponderance of endogenous over exogenous factors, had an unfavorable prognosis. Likewise, cases beginning to show symptoms on home service or on leave were poor risks. The most favorable prognoses are in the case of concussion, exhaustion, "hyperemotivity" and in the order mentioned below, the prognosis becomes increasingly poorer for conversion hysteria, gas neurosis, "neurasthenia," anxiety states, obsessional states, and effort syndromes.

Culpin points out that many front line psychiatric casualties occurred in men who had previously done excellent military service. This apparently obvious fact is often overlooked by people who expect the psychiatrist to be able to sift out practically all recruits who are likely to suffer a break-down under battle stress. Needless to say, we have no technic for forecasting neurotic reactions in persons who show no symptoms or signs of weakness in their psychic structure. Therefore, no matter how carefully we organize our psychiatric sifting devices, they will always have holes in them large enough for many potential casualties to fall through. Culpin also mentions the fact that many cases of hysteria occurred away from the front line and in home hospitals, and that these cases generally speaking went unrecognized and, therefore, further development of the illness was encouraged. This points to the great need for well-trained psychiatrists in such hospitals.

Effort syndrome, hypertension, gastro-intestinal disorders, are some of the conditions discussed. Of particular interest is the statement that, "some evidence exists that the incidence and extent of 'trench mouth' may have been influenced by emotional factors." Unfortunately, we have as yet no tests in our armamentarium by which we could detect individuals liable to such reactions.

Hargreaves' chapter on "Differential Diagnosis of the Psychoneuroses of War" offers a very helpful summary of some diagnostic problems commonly confronting those who deal with psychoneurotics. For the general medical man whose training in psychopathology has been limited to a short course of descriptive psychiatry on a Kraepelinian, Bleulerian, or allied basis, this chapter should be of inestimable value as it presents the problem from the point of view of psychodynamics. This approach is further emphasized in the next chapter by Miller on "Psychopathological Theories of Neuroses in Wartime." Too many medical men are apt to brand as culpable men who are utterly unconscious of the motives for their behavior. It should be self-evident how important this is for instance in military jurisprudence.

In Dillon's, Hadfield's, and Wright's articles, therapeutic measures of various kinds are discussed. Dillon tells of his experience with treatment in the field and points out the attending difficulties and limitations. Hadfield describes the use of suggestion and hypno-analysis in the treatment of the traumatic neuroses of war.

From Wright's article on psychotherapy the following statement is quoted verbatim because it puts before us in a few terse words the psychiatric problems of war, both social and individual:

Only if trained psychiatrists are used to their fullest capacity can the advances in psychological medicine during the last twenty years have an opportunity to prove their value. All cases of neuroses in war are not curable. Many thousand of broken, frightened men are still drawing pensions as a result. This fact must be faced squarely and honestly. It seems clear that the release of stark, primitive terror can in some cases produce irreversible changes, both psychological and physiological; and so stability is never restored.

This chapter also contains discussions of many factors of dynamic significance such as the role of suppression of harrowing experiences and its consequences to the individual.

The article of Hargreaves, Wittkower, and Wilson on "Psychiatric Organization in the Services" should receive the most careful attention of all military medical men as the statistics presented give a clear picture of the enormity of the psychiatric problem both as regards economy, and the influence on the morale of the fighting force as well as the civilian population. There is also a rather full discussion on the actual organization of the psychiatric service which is of equal importance.

The chapter on the "War of Nerves" by Bion offers a timely discussion of the sufferings of the civilian population in modern warfare.

He stresses the very important fact that the forced inactivity and lack of aim are great dangers to civilian morale and that the remedy would be one of directed activity. The reviewer is reminded of the words of Mann describing the battle scenes at Jutland (U. S. Nav. Med. Bul. 1926) where he says, talking of the medical staff during action, "It is not one's nature to sit calmly waiting for the gruesome sights which we know must come. The only remedy is work and plenty of it."

In the appendix there is much valuable information for those who would have the responsibility for carrying out the psychiatric aspects of medical service in time of war. The desirability of setting up uniform standards of nomenclature is also stressed and the reviewer would like to emphasize the obvious fact that if we are to profit from our experiences, we must have comparable data. The appendix on psychiatric pharmacology contains some suggestions for sedation and narcosis. The author states that the effectiveness of drug treatment depends more on adequate handling of the dosage than on the selection of the drug.

In conclusion, the reviewer would like to pay his respects to the authors who, in spite of the pressure of their daily work, and living in the midst of an all too real modern war, took the time to commit to writing experiences from which we can derive so much profit. For this reason alone, not to mention the purely practical ones, this book stands out as a document of the foresight, the courage, and the high culture of the British people.

NEED FOR FLIGHT SURGEONS

As a result of the aeronautical expansion, the need for flight surgeons has been tremendously increased. To meet these demands, selected medical officers will receive training at the School of Aviation Medicine at Pensacola, Florida. The course of training consists of approximately 2 months of academic instruction in the subjects relating to aviation medicine, 2 months of indoctrinal flight training and 1 month of practical instruction. Medical officers completing this training receive the designation, "Naval flight surgeon."

The training of Naval Reserve medical officers is limited to the 2 months of academic instruction, leading to the designation, "Qualified aviation medical examiner."

It is the intention to convene the next class consisting of medical officers of the regular service, for training at Pensacola, on or about August 15. This instruction will be restricted to officers of the rank of lieutenant commander or below.

It is desired that medical officers interested in this training submit their applications to the Bureau of Medicine and Surgery for consideration.

BOOK NOTICES

Publishers submitting books for review are requested to address them as follows:

The Editor,

UNITED STATES NAVAL MEDICAL BULLETIN,
Bureau of Medicine and Surgery, Navy Department,
Washington, D. C.

(For review)

THE CYCLOPEDIA OF MEDICINE, SURGERY AND SPECIALTIES, by *George Morris Piersol, B.S., M.D., editor-in-chief*, and *Edward L. Bortz, M.D., A.B., Assistant editor*. Fifteen volumes, each of which contains approximately 1,000 two-column pages; one additional index volume. F. A. Davis Co., Philadelphia, publishers, 1940. Price \$135.

This fifteen-volume work is essentially a series of monographs contributed by 807 physicians from the faculties of 87 medical schools. This is the second edition of the work. The first volume of the 12 volume edition was published in the fall of 1932. The twelfth and final volume of the original edition was published in January 1935. There were a number of reprintings of the first edition and many of the original articles were revised before each reprinting of the set. Since 1937 an annual supplement, having for its purpose the supplementing and bringing up to date of all subjects, has been published each year and will continue to be published each year for both the old and new editions at a cost of \$10 per volume.

According to the publishers, "the real reason for the new edition was not with the idea of selling over again the subscribers to the first edition but to make it possible for the newer men in the profession to purchase a complete up-to-date cyclopedic library in one unit without the necessity of purchasing and referring to a series of supplementary volumes." The present edition is not a rehash of the original. Each article was thoroughly revised and in most cases entirely rewritten and in every instance reset. It seems to this reviewer that the reasons given for publishing this second edition will hold true in a few years for a third edition.

As in any work with so many contributing authors, conflicting opinions are expressed and in many instances there is a duplication of material. It seems superfluous to have two separate discussions of the same disease. For example, in volume 9, page 750, Nathan S. Schlezinger discusses epidemic cerebrospinal meningitis; while on

page 762 J. E. Jordan discusses meningococcus meningitis; furthermore, to find a discussion of the use of sulfapyridine in this condition, one must turn to volume 14, page 476. Also in volume 9, page 760, there is a discussion of acute lymphocytic choriomeningitis while on page 780 one finds an article on acute lymphocytic meningitis, both articles covering the same subject. In volume 9, page 482, the only mention made of the use of the sulfonamide compounds in the treatment of lymphogranuloma inguinale is the following: "Sulfanilamide has been used." This subject is discussed, however, in the article "Sulfanilamide and Allied Compounds" in volume 14, page 476.

In volume 11, there is an article on protozoal diseases by Dr. John Lemuel Jelks in which this author apparently attributes pellagra to flagellate infestation, and one receives the impression that he considers pellagra to be contagious. Also, in this article the statement is made that all amebae containing 4 nuclei are pathogenic; *Endolimax nana* are described as having 4 nuclei.

There are many excellent monographs by men outstanding in their respective fields. The section on pulmonary diseases is particularly good and the section on cardiovascular diseases is excellent. However, it is considered that the attempt to compress a cyclopedia of medicine, surgery, and the specialties between the confines of 15 volumes, with subjects arranged in alphabetical order, has resulted in many cases in too much condensation, scattering, repetition, and incompleteness of information. This, plus the initial cost, the yearly payment of \$10 for the supplementary volume, together with the probability of the release of a third edition in a few years, seems to make this set a questionable investment for use other than for reference purposes in large medical libraries.

REPORTS ON MEDICAL PROGRESS, 1939, as Published in the New England Journal of Medicine. Compiled and edited by Robert N. Nye, M.D. Little, Brown and Co., Boston, Mass., publishers, 1940. Price \$5.

The subjects of these progress reports cover the specialties and the commonly encountered aspects of general medicine and surgery. The reports, as stated by one of the authors, are selective and critical rather than comprehensive. A reasonable number of references are included. However, the publication does not constitute a "year book" as each contributor summarizes the present-day ideas relative to the subject discussed without attempting to separate advances made during the year 1939. Practical rather than theoretical considerations are stressed. The volume contains much authoritative and up-to-date information concerning the diagnosis and treatment of disease and is well worth careful reading, not only by the general practitioner but also the specialist who wishes to keep in contact with recent advances in fields of medicine other than his own.

THE PRACTICE OF MEDICINE by *Jonathan Campbell Meakins, M.D., LL.D.*, professor of medicine and director of the department of medicine, McGill University; physician-in-chief, Royal Victoria Hospital, Montreal; formerly professor of therapeutics and clinical medicine, University of Edinburgh. Fellow of the Royal Society of Edinburgh; fellow of the Royal Society of Canada; fellow of the Royal College of Physicians, London; fellow of the Royal College of Physicians, Edinburgh; honorary fellow of the Royal College of Surgeons, Edinburgh; fellow of the Royal College of Physicians, Canada; fellow of the American College of Physicians. Third edition; 1,430 pp., 562 illustrations, including 48 in color. C. V. Mosby Co., St. Louis, Mo., publishers, 1940. Price \$10.

This is a rather large and comprehensive volume on medicine by one of the outstanding internists of North America. The quality of the book is very good. The printing is on excellent, slightly green tinted paper to make it easier on the eyes in reading. There are many excellent, selected illustrations with x-ray pictures and some very good colored plates distributed throughout the book.

Special chapters on diseases of metabolism, diseases of the ductless glands, diseases of the nervous system, and diseases of the urinary system have been written by distinguished specialists. There are chapters on diseases due to abnormal environments and diseases due to chemicals and drugs. Complete lists of references follow nearly every chapter. Active immunization against tetanus with toxoid injection is merely mentioned.

PHYSICAL DIAGNOSIS by *Ralph H. Major, M.D.*, professor of Medicine in the University of Kansas. Second edition, revised, 464 pages, 437 illustrations. W. B. Saunders Co., Philadelphia, publishers, 1940. Price \$5.

This is the second edition of a fine textbook devoted entirely to physical diagnosis and does not wander astray in other fields. It should be equally valuable to the practitioner and student. There are numerous well-chosen illustrations throughout the book. Many are the classic descriptions by early observers quoted with due credit given to each one.

All 26 chapters are interesting, particularly the introductory chapter and those on the pulse and history taking. In chapter V the reviewer found the author's discussion of tactile fremitus and vocal fremitus to be confusing. With this exception, the text throughout is clear-cut.

THE NEUROSES IN WAR by several authors under the editorship of *Emanuel Miller, M.A. (Cantab), M.R.C.P., D.P.M. (Cams)*, with a concluding chapter by *H. Crichton-Miller, M.D., F.R.C.P.* The Macmillan Company, New York, publishers, 1940. Price \$2.50.

This volume is reviewed in considerable detail in this issue, see Notes and Comments.

ELMER AND ROSE PHYSICAL DIAGNOSIS, revised by *Harry Walker, M.D., F.A.C.P.*, associate professor of medicine, Medical College of Virginia, Richmond, Va. Eighth edition completely revised, 792 pages, 295 illustrations. The C. V. Mosby Co., St. Louis, 1940. Price \$8.75.

This textbook of physical diagnosis is divided into two parts. Part I is devoted to the technic of physical examination and normal physical diagnosis. Part II deals with the physical diagnosis of disease. One section of the second part covers special diagnostic procedures such as electrocardiography, bronchoscopy and gastroscopy, basal metabolic rate, circulation time, tests for cardiac functional capacity, etc. Many minor as well as a few major changes have been made in order to have the subject matter conform as nearly as possible to the generally accepted present-day opinions.

The subject matter is well arranged and is presented in a clear and concise manner. The illustrations, on the whole, are well chosen. The necessity for careful and thorough physical examination of the patient in this day of numerous refined diagnostic aids, such as instruments of precision and laboratory procedures, is properly stressed.

This book should prove to be very valuable to undergraduate students and those engaged in their instruction.

PRINCIPLES OF HAEMATOLOGY by *Russell L. Haden, M.A., M.D., chief of the medical division of the Cleveland Clinic, Cleveland, Ohio; formerly professor of experimental medicine, in the University of Kansas School of Medicine, Kansas City, Kansas.* Second edition; 362 pages. Illustrated. Lea & Febiger, Philadelphia, 1940. Price \$4.50.

Like the first edition published in 1939, the second covers the usual technical hematological methods as well as the important diseases which manifest themselves in the blood. The errors usually present in a first edition of any book have been corrected. The additions are few, the most useful of which are the technic of blood typing and a very short discussion of the role of vitamin "K" in hemorrhagic conditions. There are many good illustrations and diagrams in black and white. The diagrams concerning the mechanisms of blood production, of anemias of polycythemia, of leucocytosis, of leukopenia and clotting are most ingenious and they help greatly in the gaining of a concept of these conditions.

The book is excellently printed and is recommended to every student of medicine, undergraduate or graduate.

INTRODUCTION TO MEDICINE for Schools of Nursing by *Don C. Sutton, M.S., M.D., associate professor of medicine, Northwestern University School of Medicine; attending physician, medical division of the Cook County Hospital; chief of the cardiac clinic, Cook County Hospital, Chicago; attending physician, Evanston Hospital.* With introduction by *Ada Belle McCleery, R.N., superintendent, Evanston Hospital, Evanston, Illinois.* 642 pages with 144 text illustrations and 14 color plates. C. V. Mosby Co., St. Louis, publishers. 1940. Price \$3.25.

This is really a compend of the practice of medicine, each subject being treated very briefly. The intention is to give the nurse a bird's-eye view of the field of human diseases, their causes and treatment.

A **TEXTBOOK OF MEDICAL DISEASES FOR NURSES**, Including Nursing Care, by *Arthur A. Stevens, A.M., M.D.*, formerly professor of applied therapeutics in University of Penna. and of therapeutics and clinical medicine in Women's Medical College of Penna.; honorary consulting physician to Phila. General Hospital; consulting physician to St. Agnes Hospital, Philadelphia, etc., and *Florence Anna Ambler, R.N., A.M.*, director of nursing service and principal of school of nursing, Children's Hospital, Pittsburgh, Penna.; formerly educational director, Philadelphia General Hospital, and principal, School of Nursing, Samaritan Hospital, Troy, N. Y. 4th edition, revised. 551 pages. W. B. Saunders Co., Phila., publishers, 1940. Price \$2.75.

This book is virtually a compend of medicine and though necessarily very brief, both nervous diseases and skin conditions are included. In discussing treatment, emphasis is placed on the nursing care.

PHYSICAL THERAPY FOR NURSES by *Richard Kovacs, M.D.*, clinical professor and director of physical therapy, New York Polyclinic Medical School & Hospital; attending physical therapist, Manhattan State, Harlem Valley State and West Side Hospitals; visiting physical therapist, New York City Department of Correction Hospitals; consulting physical therapist, New York Infirmary for Women and Children; *Mary Immaculate Hospital, Jamaica, N. Y., Hackensack Hospital, Hackensack, N. J.* 335 pages, 99 engravings. Lea & Febiger, Phila., Penna., publishers, 1940. Price \$3.25.

This is an extremely handy and practical little book for nurses and technicians in physical-therapy departments.

NEOPLASTIC DISEASES, A Treatise on Tumors, by *James Ewing, A.M., M.D., Sc.D., LL.D.*, professor of oncology at Cornell University Medical College, New York City, consulting pathologist, Memorial Hospital. Fourth edition, revised and enlarged with 581 illus. 1160 pages. W. B. Saunders Co., Philadelphia, publishers, 1940. Price \$14.

For two decades this great treatise on tumors has been indispensable as a reference work for pathologists. Twelve years have elapsed since revision, a period that has brought many additions to our knowledge of neoplastic diseases. To critically review the multitude of these contributions not only in general pathology and clinical medicine, but in the allied sciences of physics, chemistry, physiology and genetics and to collect the essential facts, is a task well and thoroughly accomplished in this revision. The general arrangement of the book has not been changed and much old material has been deleted. The microphotographs in black and white are numerous and good. A very extensive bibliography is found in the back of the book. Ewing's *Neoplastic Diseases* will continue to be our outstanding reference book on tumors.

A **TEXTBOOK OF PATHOLOGY**, by *W. G. MacCallum*, professor of pathology and bacteriology, Johns Hopkins University, Baltimore, Md. Seventh edition, thoroughly revised, 1302 pages, 697 illus. W. B. Saunders Co., Philadelphia, publishers, 1940. Price \$10.

This well known standard textbook of pathology has long been a favorite in medical schools. It was first published in 1916 and has been kept up to date since by careful revision every 4 years. In this latest revision, as in former years, the same plan of arrangement and high standard of subject matter and illustrations have been maintained. A well-chosen bibliography is given at the end of each chapter. It continues to be a leading textbook of pathology.

MAMMALIAN GENETICS by *William E. Castle, professor emeritus of genetics, Harvard University, research associate in genetics, University of California.* First edition; 169 pages, illustrated, Harvard University Press, Cambridge, Massachusetts, 1940. Price \$2.

Professor Castle outlines in a clear and systematic manner the basic principles of heredity, most of which can be verified by breeding experiments with mammals. The book is well illustrated by 131 photographs and drawings in black and white and 8 tables which aid materially in the visualization of the effects of cross mating. This volume will serve excellently as a text book in the teaching of genetics as well as a reference book for others interested in the field of hereditary influences.

TRENDS IN NURSING HISTORY by *Elizabeth Marion Jamieson, B.A., R.N., and Mary Sewall, B.S., R.N.* W. B. Saunders Co., Phila., Penna., publishers, 1940. Price \$3.

This is a book that deserves more than passing notice for it is at once a scholarly and readable work on a long and most important chapter of medical history. Except for some overemphasized material on Florence Nightingale, the real history of nursing has been overlooked. This book is not definitive but it is the most complete this reviewer has seen on this subject. As the writers point out "nursing has been identified with woman's life and work since the beginning of time." They show the relationship of the fact to social development and the contribution made by women to it. The general history given to form the background is particularly good. This book should bear to the history of nursing the place that Garrison's text forms to the history of medicine.

THE EMPEROR'S ITCH by *Reuben Friedman, M.D., assistant professor of dermatology and syphilology, Temple University School of Medicine, Philadelphia.* 82 pages, 10 illustrations. Froben Press, Inc., New York, publishers, 1940. Price \$1.50.

Doctor Friedman, in writing this very interesting monograph, has, in a scientific way, exploded a generally accepted theory that Napoleon suffered throughout his adult life with scabies. Although "the little corporal" may have at one time had a scabetic infection, the author is convinced that dermatitis herpetiformis was the real cause of the severe pruritus so troublesome during his adult life.

Although of no major historical value, this little book can be recommended as an unusual addition to Napoleonic lore.

BUNDY'S ANATOMY AND PHYSIOLOGY revised and edited by *S. Dana Weeder, M.D.*, former instructor in anatomy, University of Pennsylvania; surgeon, Germantown Hospital; associate surgeon, Chestnut Hill Hospital, Philadelphia, Pa. 7th ed. 490 pages. The Blakiston Co., Philadelphia, publishers, 1940. Price \$2.75.

This is a complete revision of an old standby for use as a text in high schools and colleges. Among the features of this edition are a number of valuable anatomical tables and a good glossary. The illustrations and binding are particularly good.

APPLIED MICROBIOLOGY AND IMMUNOLOGY FOR NURSES by *Charles F. Bolduan, M.D.* and *Nils W. Bolduan, M.D.* W. B. Saunders Co., Philadelphia, Penna., publishers, 1940. Price \$2.25.

Dr. Charles Bolduan is Director of the Bureau of Health Education in New York City and has had an extensive experience with the training of nurses and technicians. The fact that this work has gone through 8 editions attests in some measure to its excellence. This review adds new features regarding salmonella, moulds, protozoa, rickettsia and viruses.

THE SURGERY OF THE ALIMENTARY TRACT, by *Sir Hugh Devine, M.S., F.R.A.C.S.*, F.A.C.S. formerly senior surgeon, St. Vincent's Hospital Clinical School, Melbourne, and other Stewart lecturer in surgery, Melbourne University; president of Royal Australasian College of Surgeons; honorary fellow of the Association of Surgeons of Great Britain and Ireland; chairman, editorial committee of the *Australasian and New Zealand Journal of Surgery*. Pp. 1046, illustrated. Williams & Wilkins Co., Baltimore, Md., publishers, 1940. Price \$15.

The author states that in writing this book he had no intentions of producing a comprehensive treatise on abdominal surgery but rather to write upon the subjects that his long experience as a clinical teacher and general surgeon so well fit him. In it he attempts to bring us pictures of living pathology as seen at the operating table and to fit these pictures into clinical, radiological, and other forms of surgical diagnosis.

The work is divided into 4 parts and 93 chapters. Part I comprises 32 chapters devoted to the diagnosis of surgical dyspepsia. His reason for spending so much time on diagnosis is that in his opinion clinical diagnosis has not kept pace with radiological diagnosis and surgical treatment. This lagging in clinical diagnosis has frequently been the cause of patients reaching the surgeon too late. Many short-case histories are cited to bring out some diagnostic feature. In these chapters he attempts to show the mechanism of causation of dyspepsia and why the same disease may produce many varieties of dyspeptic patterns, and how the disease in one case may produce dyspepsia and in another be silent.

Part II contains 47 chapters and deals with the surgical treatment of diseases which give rise to dyspeptic syndromes. It also deals with those organs in the abdomen other than the stomach, gallbladder, pancreas, and spleen, which may cause some dyspeptic manifestations. In these chapters he gives in detail the technic of the various operations. This is especially so in gastric surgery for which he has long been noted. He places great emphasis on gentleness in handling of organs and for that reason advocates the use of his self-retaining operating frame which makes it possible for him to operate upon abdominal organs in their natural site rather than dragging them out onto the abdominal wall. A most interesting chapter deals with hydatid disease, which is apparently very common in Australia and New Zealand. He states "evidence of an enlarged liver in a patient in good health is almost certain to be due to this cause." The second section deals with postoperative complications in a thorough manner.

Part III covers abdominal emergencies which may involve the upper and lower abdomen and chiefly has to do with acute perforation of a hollow organ and intestinal obstruction.

Part IV is mostly concerned with surgery of the appendix, large bowel, and rectum. These chapters are in keeping with the rest of the book. Many American surgeons will not agree with his views on abdominal drainage in acute appendicitis that: "The abdominal cavity (and the abdominal wound) should as a routine be drained."

He emphasizes the fact that the operation of partial gastric exclusion, which in this country is frequently spoken of as the Devine operation, is not designed for routine use but for special application in certain cases, and that in selected cases no other operation can attain its possibilities both as regards lessened risks and permanent cure. In his experience this operation has been found to be practically free from any of the unpleasant after effects that often attend gastro-enterostomy.

The author's aim throughout the book is to further the reader's diagnostic ability and to facilitate surgery, both of which tend to reduce the morbidity and mortality rates.

This work will not displace the usual textbooks on the subject but it will be a valuable additional source of knowledge for the post-graduate student and general surgeon.

The text is very well illustrated and the index is adequate. No attempt is made to give an extensive bibliography.

PRINCIPLES OF SURGICAL CARE, SHOCK, AND OTHER PROBLEMS, by *Alfred Blalock, M.D., professor of surgery, Vanderbilt University School of Medicine, Nashville, Tennessee.* Pp. 325, Ill. 13. C. V. Mosby Co., St. Louis, publishers, 1940. Price \$4.50.

This book deals in the main with the general principles of the preoperative and postoperative treatment of surgical patients. The

author emphasizes the fact that the operation is only part of the treatment of surgical patients. There is no question that decreases in the morbidity and mortality rates are due more to the preoperative and postoperative treatment than to any other factor.

The book deals primarily with shock, its cause, prevention, and treatment. The author, whose name has been closely linked with the subject, speaks authoritatively.

This is an excellent book, well written and practical. The references are complete.

The book is highly recommended to those in the profession called upon to treat shock from whatever cause.

THE RECTUM AND THE COLON, by *E. Parker Hayden, A.B., M.D., F.A.C.S.*, assistant in surgery in Harvard Medical School, Boston, Mass., assistant surgeon and chief of rectal clinic of Massachusetts General Hospital, Boston, Mass. 434 pages. illus. Lea & Febiger, Philadelphia, publishers. Price \$5.50.

The author presents subject matter which in many instances is a tug of war of opinion between the internist and surgeon. One can see that the author primarily covers his subject from a surgical point of view, but his conservatism in dealing with various problems of diseases of the rectum and colon, leaves no place for objection on the part of the internist.

One is apt to disagree with the author on usage of mineral oil by mouth to produce soft stool, as it is known at present that mineral oil *per se* is an irritant to rectal mucosa.

Numerous chapters have analytical and statistical studies of the discussed disease, a very welcome addition for reference work. The chapter on irritable colon shows recent speculations and experiments on reflex of defecation and expresses latest opinion that the term colitis is inappropriate when applied to a syndrome of this type, which is only an abnormal physiological response of the colon and rectum to a stimulus originating elsewhere than in the bowel itself.

The chapter on ulcerative colitis is excellently presented. It is conservative and views the administration of Bagen's vaccine with understanding skepticism. The author also quotes from Massachusetts General Hospital cases which suggest relationship between intake of cascara and causation of melanosis coli, a relationship suggested also by Bockus, Buie, and others.

The book is written in a concise manner and is a useful addition to a reference shelf.

OPERATIVE SURGERY by *J. Shelton Horsley, M.D., LL.D., F.A.C.S.*, attending surgeon, St. Elizabeth's Hospital, Richmond, Va., and *Isaac A. Bigger, M.D.*, professor of surgery, Medical College of Virginia, surgeon-in-chief, Medical College of Virginia Hospitals, Richmond, Va., with contributions by *C. C. Coleman, M.D., F.A.C.S.*; *John S. Horsley, Jr., M.D.*, *Austin I. Dodson, M.D., F.A.C.S.*, *Donald M. Faulkner, M.D.*; with illustrations by *Helen*

Lorraine. Fifth edition. In 2 volumes. 1567 pages, 1391 illustrations. C. V. Mosby Co., St. Louis, Mo., publishers, 1940. Price, \$18 a set.

The fact that the fifth edition of this work is appearing only 3 years after the fourth, is ample proof of the popularity of the work. The advances in surgery have been so rapid in the past few years that the authors found it necessary to practically rewrite many of the chapters. In producing this work the authors have been ably assisted by other members of the staff of the Medical College of Virginia Hospitals.

It is impossible to point out all of the outstanding features of a work of this size. Vol. I contains 41 chapters and 768 pages; vol. II, 36 chapters and 798 pages. From the beginning to the end there is a definite attempt to bring the subjects under discussion up to date. The chapters on blood banks, chemotherapy, and heart surgery are particularly new and very good. There has been no attempt to produce an encyclopaedic work on surgery, the authors being content in describing operations that have proved to be the most successful in their hands.

A limited bibliography follows each chapter. The printing and binding are both excellent. The text is well illustrated and the index is comprehensive. The detailed description of operations in conjunction with the excellent illustrations will make this work a special favorite of the occasional surgeon.

SURGERY OF THE HAND, Wounds, Infections, and Closed Traumata, a Book for the Practitioner and the Surgeon by *Marc Iselin, M.D., Surgeon, The American Hospital, Paris.* Translated by *T. M. J. d'Offay, M.B., Ch.B. (Edin.), F.R.C.S. Eng., surgeon and deputy medical superintendent, City General Hospital, Leicester;* and *Thomas B. Mouat, M.D., Ch.M. (Edin.), F.R.C.S. Eng. Surgeon, The Royal Infirmary, Sheffield, lecturer in Surgery, The University of Sheffield.* 353 pages. 135 illustrations, including 8 plates. The Blakiston Company, Philadelphia, Publishers, 1940. Price, \$5.50.

Although this book has gone through two previous French editions this is the first time it has been translated into the English language. The work is divided into four parts dealing with wounds, infections of the hand, closed traumata of the hands and fingers, and assessment of incapacity.

The author points out the great frequency of wounds of the hands and fingers. In some clinics this type of accident accounts for as high as 40 percent of all injuries. The percentage of infected wounds is also reported as high as 21 percent. He cites statistics published by the staff of the teaching hospital at Zurich (1920-1930) which showed wounds of the finger caused the greatest mortality (17) while compound fractures of the femur were second with 7 deaths.

He is of the opinion that this type of wound is too frequently neglected due to the fact that the young surgeon is more interested in

abdominal surgery and the older experienced surgeon is reluctant to make the effort to keep abreast with a branch of surgery which is considered to be minor in nature.

Every wound of the finger should receive similar treatment to that given wounds in other parts of the body, all aseptic precautions should be taken, the patient must be anaesthetised, the lesion explored, contused tissue excised, loose fragments of bone removed, fractured ends of bones trimmed, and the wound must be stitched only if conditions are favorable for primary union.

The treatment of every conceivable type of injury of the hand and fingers is described and many short case histories are interspersed to bring out important features.

The printing and binding are excellent and the text is well illustrated. The references are mostly foreign and are not extensive. The index is adequate.

This is an extremely valuable book for anyone doing surgery and especially for the industrial surgeon.

GYNECOLOGICAL AND OBSTETRICAL PATHOLOGY With Clinical and Endocrine Relations, by *Emil Novak, A.B., M.D., D.Sc. (Hon. Dublin) F.A.C.S., associate in gynecology, The Johns Hopkins Medical School; gynecologist, Bon Secours and St. Agnes Hospitals, Baltimore; fellow, American Gynecological Society, American Association of Obstetricians, Gynecologists and Abdominal Surgeons and Southern Surgical Association; honorary fellow, Royal Institute of Medicine, Budapest; Sociedad d'Obstetricia et Ginecologia de Buenos Aires; Central Association of Obstetricians and Gynecologists, Texas State Assoc. of Obstetricians and Gynecologists; past chairman, Section on Gynecology and Obstetrics, American Medical Association.* 496 pages with 427 illustrations. W. B. Saunders Co., Philadelphia, publishers, 1940. Price, \$7.50.

In this book of 496 pages, Doctor Novak has apparently struck the happy medium of presenting his material so as to hold the interest of the student and in sufficient detail to give the pathologist and specialist what they seek.

The publishers have done their customary excellent job of book-binding and printing on good paper. The illustrations are abundant and surprisingly superior. These are largely photomicrographs from carefully selected, well prepared, representative histologic sections which add much to the instructiveness and usability of the book.

The author, one of the outstanding authorities in this field, has utilized a natural, engaging, narrative style which makes the technical material pleasant reading even for the novice. It is apparent, in spite of a brief but well chosen bibliography, that Dr. Novak writes from the background of a tremendous personal experience and has himself made contributions to many of the subjects covered in the various chapters of his book.

Four of the 24 chapters in the book are devoted to pregnancy. One of these describes the process of migration of the fertilized ovum, implantation and placentation as it is known today. The chapter on hydatidiform mole and chorionepithelioma malignum is especially good. The author stresses the difficulties occasionally involved in making a correct diagnosis and particularly the pitfalls of diagnoses based on material obtained by curettage. His attitude toward the so-called malignant hydatidiform mole is well and conservatively presented. The chapter on abnormalities and diseases of the placenta by L. M. Hellman, is concise, contemporary, and among other things gives a brief but critical appraisal of the histologic diagnosis of placental syphilis and its probable confusion with erythroblastosis on the basis of former criteria.

The general plan of the remainder of the text is to introduce each anatomical subdivision with a brief consideration of its anatomy and normal anatomical physiology before considering its pathology. So we find instructively presented how hormonal influences produce variable histologic pictures in the ovary, endometrium and the fallopian tube. Some may feel that too much space is given to the latter.

It is refreshing to note the author's ability to present debatable issues fairly and critically and his avoidance of dismissing with a dogmatic statement a border-line problem which only further study will solve.

On page 6, the term "dehiscence" is obviously a typographical error of which there are few. Biopsy is a term frequently misused to signify the obtaining of tissue for diagnosis. It would be helpful to describe in more detail the histology and to show some higher magnification of the lesions of granuloma inguinale particularly some showing the Donovan bodies. Mention of the use of the silver impregnation method to demonstrate these bodies would be informative to those using the text as a reference. Obviously, these criticisms in no way detract from the fundamental excellence of the text. Addition, here and there, of similar detail in small type in subsequent editions will not materially increase the size of the book or lessen its attractiveness.

The chapters on the pathology of the endometrium including adenocarcinoma of the corpus uteri are well done. The author's skeptical attitude toward the rôle of myoma as a predisposing factor in the development of adenocarcinoma of the uterus is wholesome and realistic. It should help eventually to lessen the wholesale sacrifice of the small myomatous uterus whose only offense often is its apparent flirtatious challenge to the susceptible and uncritically aggressive surgeon.

Chapters 18 to 29 are given over to the ovary and ovarium pathology. While this may appear as a top-heavy division of material, it does not prove to be so. Dr. Hovak has handled this difficult subject

critically, interestingly, and open mindedly. He points out some of the difficulties of diagnosis in this field and the impossibility at times of making an absolute differentiation. This, obviously, is as it should be. It is an attitude that permeates the text and gives it the stamp of authenticity. His discussion of unsettled theoretical aspects of these tumors is brief, to the point, and yet sufficiently full to give the reader a good appreciation of the problem involved. The discussion of Krukenberg tumors is excellent.

Throughout the text clinical and pathologic correlations, where applicable, are made. These obviously add to the value of the text for the clinician and student and yet do not detract from its fundamental character as a book of special pathology. One feels no hesitation in recommending this volume as an authoritative text for which there should be a real demand. It will be appreciated by students studying gynecologic pathology, by teachers of gynecology for whom it will serve as a compact unified reference, and by practitioners interested in gynecology. Pathologists, who are often confronted by special problems in gynecological pathology, will find it an invaluable source of help.

MANUAL OF MEDICAL AND SURGICAL EMERGENCIES, edited by *J. C. Geiger, M.D.*, director, department of public health, City and County of San Francisco, California. Pp. 199. J. W. Stacey, Inc., San Francisco, publishers, 1940. Price \$2.50.

This small manual is the outgrowth of the needs for specific information on emergency hospital practice. More than 50 specialists, either actively connected with the service of the San Francisco Emergency Hospital at this time or formerly associated with it, have contributed to the work. It not only covers practically every medical and surgical emergency that might arise but also deals with regulations of an emergency hospital service and the various legal problems that are common to such practices.

For a book of its size it contains a world of common-sense information. Owing to the limited space there is little controversial discussion; what one gets mostly is bold-faced facts. It contains no illustrations or bibliography but has an adequate index. It would be a handy book in any doctor's library.

BONE GRAFT SURGERY IN DISEASE, INJURY AND DEFORMITY by *Fred H. Albee, M.D., LL.D., Sc.D., F.A.C.S., F.I.C.S.*, president, International College of Surgeons; past president, American Orthopedic Association; chairman, Rehabilitation Commission of the State of New Jersey. Assisted by *Alexander Kushner, M.D., B.Sc.* 403 pages, illustrated. D. Appleton-Century Co., New York, publishers, 1940. Price \$7.50.

In this book the pioneer of bone graft surgery boils down his experiences of 30 years and presents them in a clear, concise, and convincing manner. He spends little time in discussing controversial matters and is content in describing not only the many operations that he has

personally originated but also those operations developed by other experienced surgeons which have stood the test of time.

He states that the outstanding reasons for the rapid development of bone graft surgery in such a short time is due to development of the x-ray, the bone mill, the orthopedic traction table, and to ample proof that autogenous bone could be transferred from one part of the body to the other.

The book is small, containing only 9 chapters and 403 pages. However, it contains practically all of the information that exists on the subject at the present time. The opening chapter on the general principles of bone grafting gives one an insight as to the reason for the author's outstanding success in this line of work. From his follow-up study of more than 6,000 cases he is positive in stating that when a bone graft is properly placed in a favorable environment there is no question but that it lives and reacts according to the law laid down by Wolff.

The printing and binding are excellent and the text is exceptionally well illustrated. There is a limited bibliography following each chapter and a good index of both authors and subjects.

PHYSICAL TREATMENT BY MOVEMENT, MANIPULATION AND MASSAGE by *James B. Mennell, M.A., M.D., B.C. (Cantab.), etc., consulting physiotherapist, St. Thomas' Hospital, and lecturer, Massage Training School.* Fourth edition. 669 pages with 281 illustrations including 32 plates, 8 in color. The Blakiston Co., Philadelphia, Publishers, 1940. Price \$7.

This volume, the fourth edition of a work first published in 1917, provides a valuable treatise for those who have to do with massage and kindred aspects of physiotherapy and also for the medical profession in general.

It starts with a good account on massage, its types, effects, technic, etc. Relaxed (passive) movements are fully considered, and a good tabulation of objectives is provided. Active movements are next considered under various subdivisions as to type and as applied to various parts of the body. Various kinds of mechanical apparatus are described and shown in illustrations. In general the illustrations are abundant and well chosen.

The rest, and greater part of the book, is given over to the application of these various modes of treatment to a great variety of conditions; fractures, sprains, muscle injuries, reeducation are all considered. Various neurological psychiatric conditions are also taken up. The author's viewpoint and recommendations appear sensible and conservative.

Backache is given an especially thorough and helpful analysis. The more common causes are taken up and in addition, the claims and theories of the various irregular schools of practice are gone into and properly evaluated.

In general, this is an excellent volume that can be dipped into with profit by nearly all of us. We all tend to neglect massage and manipulative treatment perhaps because largely we lack time and also because good masseurs are few and far between. Thus a book such as this may well serve as a useful reminder as well as a work of reference.

TEXTBOOK OF OPHTHALMOLOGY, Vol. III, Diseases of the Inner Eye, by Sir W. Stewart Duke-Elder, M.A., D.Sc. (St. And.), Ph.D. (Lond.), M.D., Ch.B., F.R.C.S., surgeon oculist to H. M. The King; colonel A.M.S., consulting ophthalmic surgeon to the British Army, ophthalmic surgeon, St. George's Hospital; honorary research associate, University College, London; lecturer in ophthalmology, St. George's Hospital Medical School, University of London, etc. Pp. 1353, with 1,140 illustrations, including 164 in color. C. V. Mosby Co., St. Louis, Publishers, 1940. Price \$18.50.

This third volume of a monumental text on ophthalmology deals with diseases of the inner eye. Volume I dealt with the development, forms, and function of the visual apparatus. Volume II dealt with the clinical methods of examination, congenital and developmental anomalies, general pathological and therapeutic considerations, and diseases of the outer eye.

In dealing with diseases of the inner eye the text considers the following subdivisions: (a) The uveal tract, (b) the retina, (c) the optic nerve, (d) the lens, (e) glaucoma and hypotony, and (f) intraocular parasites.

This text is so comprehensive it is encyclopedic. Thirty pages alone are devoted to the etiology of uveitis. All subjects are likewise treated. At the beginning of each chapter, Duke-Elder picks out some outstanding ophthalmologist and includes a brief biography and picture. For example, chapter 33, devoted to diseases of the optic nerve, is prefaced by a biography and picture of the late renowned George Edmund De Schweinetz, who is rightly placed at this point because of his last outstanding contribution—his Bowman's lecture in 1923, which dealt with the optic nerve.

So complete is the text that references are made and dealt with in detail as for instance, the earliest known writings on cataract by Susruta, the great surgeon of ancient India 1,000 years B. C. This text is so replete that all the literature, for instance, in deficiency cataract, is included, viz, the effect of depletion of certain of the amino-acids, tryptophane and cystine in the production of cataract. The absence of Vitamin B₂ (lacto-flavin) in the production of cataract and its uses in prevention is included. Literature up to 1939 is incorporated.

The internist and those medical officers interested in tropical medicine will find the chapter on intraocular parasites worthwhile for reference.

OFFICE UROLOGY, With a Section on Cystoscopy, by *P. S. Pelouze, M.D.*, *assistant professor of urology, University of Pennsylvania, consulting urologist, Delaware County Hospital; special consultant to United States Public Health Service; member of Board of Directors, American Social Hygiene Association and American Neisserian Medical Society.* Pp. 766 with 443 illustrations. W. B. Saunders Co., Philadelphia, Pa., publishers, 1940. Price \$10.

In the library of the naval practitioner this volume would represent a valuable asset, for it presents a concise and readable survey of our most important specialty. The reputation of Pelouze is well established, and, to judge from this latest work, justly so.

Divided into three convenient sections, this book is most useful to the urological novice. Three hundred pages present a thorough discussion of anatomy and physiology, diagnostic measures, and methods of treatment. An equal space is given to a routine presentation of diseases of the genito-urinary system, not omitting a brilliant discussion of the sexual problem; and a long but agreeable meander through the urinary tract with cystoscope and x-ray. This last is generously illustrated, but probably of interest only to the experienced cystoscopist.

This is not an all-inclusive work, but in one respect it is distinctively useful. More than any author, Pelouze is a teacher of the uninitiated. Instruments are described, an office is equipped, and technics are explained in detail.

In the treatment of gonorrhea, Pelouze reminds many of us that cures were obtained before the use of the sulfonamide drugs, and he emphasizes the present-day importance of local treatment. Five days, he insists, is an adequate trial period for the oral route of therapy; improvement failing, it should be abandoned in favor of local treatment. In this latter he finds use for little beyond a weak (5 percent) solution of mild protein silver and a 1:8000 solution of the permanganate of potash. He watches the urine carefully for evidence of posterior spread, and, when this occurs, ceases all local treatment. With subsidence of symptoms he begins intravesical irrigations of the permanganate of potash solution, gently and at low pressure. With improvement, but not before the lapse of 3 weeks, a course of gentle prostatic "stroking" is begun. He recommends little else.

He is perhaps, at times, too personal and too vehement for the author of a scientific work, but, since this attitude usually accompanies a passionate insistence upon meticulous method and care, it seems warranted. The most experienced may profit by his scorn of slipshod diagnosis.

CLINICAL UROLOGY by *Oswald Swinney Lowsley, A.B., M.D., F.A.C.S., director of the department of urology (James Buchanan Brady Foundation) of the New York Hospital, and Thomas Joseph Kirwin, M.A., M.S., M.D., F.A.C.S., attending surgeon of the department of urology (James Buchanan Brady Foundation) of the New York Hospital.* Drawings by *William P. Didusch.* Pp. 1,684, illustrated. Williams & Wilkins Co., Baltimore, publishers, 1940. Volumes I and II. Price, \$10 per set.

The size of this book should not frighten off the prospective reader or purchaser since it is constructed in two volumes of convenient size, and is thus much less forbidding than one might suppose from the page numbers.

The authors have intended the book "for the medical student, the general practitioner, the general surgeon, and as a reference work to the trained urologist." They evidently have listed these classes of readers in their order of importance as to the utility of the book to the reader. This reviewer believes that the order of the listing should be reversed. The book is of inestimable value to the urologist because it constitutes a highly expert source of the most modern information on technical details of urological operations. It is likewise of great value to the general surgeon who includes urological surgery in his work. The general practitioner, and still more, the medical student, would find their purposes better served by a smaller work devoted more to office methods of diagnosis and nonsurgical methods of treatment.

For those engaged in major or minor surgery of the genito-urinary tract this book could not be recommended or praised too highly. Its value is enhanced immeasurably by the drawings of Didusch who is widely acknowledged as an American master of this field of art.

A most excellent covering of the whole subject of gonorrhea is presented in a chapter of some 50 pages. This chapter is written with the collaboration of *Dr. Andrew P. Peterson* and happily contains the sanest and most modern concepts of the disease. This chapter is of definite value to the general practitioner.

DERMATOLOGY AND SYPHILOLOGY FOR NURSES, Including Social Hygiene, by *John H. Stokes, M.D., professor of dermatology and syphilology, School of Medicine, University of Pennsylvania.* 365 pages, illus. W. B. Saunders Co., Philadelphia, publishers, 1940. Price \$2.75.

Dr. Stokes, in this third edition, presents a valuable work which can be highly recommended to both student and practicing nurse. The text is readable, concise, and not too highly technical. The author has shown good judgment in the choice of the subject material, both as to extent and detail. Clinical features, diagnosis, and treatment of skin and genitoinfectious diseases are discussed in an efficient and interesting manner.

The value of the book is enhanced by the section on the principles of social hygiene and by a well-chosen glossary.

TEXTBOOK OF DENTAL ANATOMY AND PHYSIOLOGY by *Russell C. Wheeler, D.D.S., F.A.C.D., associate professor of dental anatomy, human and comparative, St. Louis University School of Dentistry; chairman of the department.* Pp. 415, with 394 illustrations. W. B. Saunders Co., Philadelphia, Pa., publishers, 1940. Price \$6.50.

This book follows rather closely the pattern of other textbooks on the same subject. Exclusive of the illustrations it does not contribute a great deal that has not been presented to the profession at some previous time. Many of the illustrations present the subject in a different manner although a few have appeared in other publications. It is a splendid, well-written, modern, and very easy-to-read textbook for the student and suitable for use in college.

THE AMERICAN TEXTBOOK OF OPERATIVE DENTISTRY In Contributions by Eminent Authorities edited by *Marcus L. Ward, D.D.Sc., Jonathan Taft professor of dentistry, School of Dentistry, University of Michigan.* Seventh edition. Pp. 925, illustrated with 587 engravings. Lea & Febiger, Philadelphia, Pa., publishers, 1940. Price \$11.

This book includes operative dentistry in its broadest aspect. Many contributors have covered the field almost too well, in that they have repeated certain phases of the subject matter. However, it is a modern, comprehensive text for the dental student and serves as a review and source of reference for the dentist. While it offers little in originality, it covers quite adequately new technics and theories presented in current literature.

THE DIVISION OF PREVENTIVE MEDICINE

Commander C. S. Stephenson, Medical Corps, United States Navy, in charge

INDUSTRIAL HYGIENE AND THE NAVY IN NATIONAL DEFENSE¹

This paper presents an outline of the administration of industrial hygiene in navy yards, which are the chief industrial units of the Navy.

INDUSTRIAL ORGANIZATION OF NAVY YARDS

The mission of a navy yard is primarily the construction, maintenance and repair of naval vessels. The central administration of navy yards and all industrial shore stations of the Navy is vested in the Assistant Secretary of the Navy, in whose office is the Shore Establishments Division of the Navy Department.

In addition to the 11 navy yards, there are the following specialized industrial plants: the plants for the building of submarines at Portsmouth, N. H., and Mare Island, Calif.; the Naval Gun Factory at the Navy Yard, Washington, D. C.; the torpedo factories at Newport, R. I., and Alexandria, Va.; the powder plant at Indian Head, Md.; the aircraft factory at Philadelphia for experimental aircraft construction and repair; the naval armor plate plant at Charleston, W. Va.; and the Naval Clothing Factory at Brooklyn.

ORGANIZATION OF THE NEW YORK NAVY YARD.—This navy yard may be taken as typical of a major yard. Its organization falls under two departments, *i. e.*, the industrial department, headed by a navy captain of the engineering branch, and an operations or military department, under the direction of a naval line captain. It may be stated that 90 percent of the activities of a navy yard are industrial.

EXTENT OF THE CIVILIAN INDUSTRIAL FORCE OF THE NAVY.—The combined industrial force of all navy yards and shore stations will probably reach 180,000 in 1941. In addition, the employee volume in the commercial naval shipbuilding plants will probably reach a peak of over 100,000.

INDUSTRIAL HYGIENE ADMINISTRATION OF A NAVY YARD

THE MEDICAL DEPARTMENT.—The medical staff of the New York Navy Yard consists of 10 medical officers, 5 dental officers, 1 nurse,

¹ Abstracted from an address presented by Captain E. W. Brown (MC), U. S. N. at the Fifth Annual Meeting, Air Hygiene Foundation of America, Inc., Pittsburgh, Pa., November 13, 1940, and published in *War Medicine*, 1:3, January 1941.

45 enlisted men, and 2 civilian clerks. The chief activities with reference to industrial personnel are as follows:

1. *Preemployment physical examination.*—All applicants for federal jobs are given a physical examination.

2. *Periodic physical examinations.*—Medical supervision of employees exposed to potential health hazards.

3. *Physical examination of employees for retirement.*

4. *Diagnosis, treatment and disposition of industrial injuries and occupational diseases.*

5. *Administration of compensation cases.*

6. *Industrial hygiene and plant sanitation.*

THE INDUSTRIAL MEDICAL OFFICER.—An officer of the medical staff of the navy yard is specifically detailed for industrial hygiene administration subject to the direction of the senior medical officer. Figure 36 outlines the scope of his activities.

1. *Adviser to the safety engineer.*—There must be close and efficient correlation of the work of the safety engineer and that of the industrial medical officer.

2. *Inspections.*—The industrial medical officer is responsible for the general supervision of plant sanitation. This necessitates frequent plant inspections.

3. *Supervision of special physical examinations.*—This includes applicants for employment who report previous exposure to potential industrial health hazards; periodic examinations of those exposed to these hazards; examination of those transferring to other shops where there is a question of occupational disability.

4. *Medical surveys of occupational groups.*

5. *Administration of the medical aspects of claims for compensation occupational disease.*

6. *Supervision of preparation of accident and occupational-disease reports for the Navy Department.*

THE SAFETY ENGINEER.—A civilian safety engineer is stationed at the Navy Department as adviser to the head of the Shore Establishments Division. A naval officer is assigned to each yard as the safety engineer.

1. *Accident and unsafe practice control.*—The safety engineer investigates all lost-time accidents to determine cause and advises measures to prevent their recurrence, such as safety devices and safety education. The Navy Department publishes the comparative safety scores of all navy yards monthly. This has developed a keen competition among employees. A great advance has been made by the Navy Department in accident reduction, beginning with an intensive safety campaign in navy yards in 1926. In a 12-year period since 1926, the accident rate was lowered practically one-half; the severity rate reduced from 2.2 per year to 0.5.

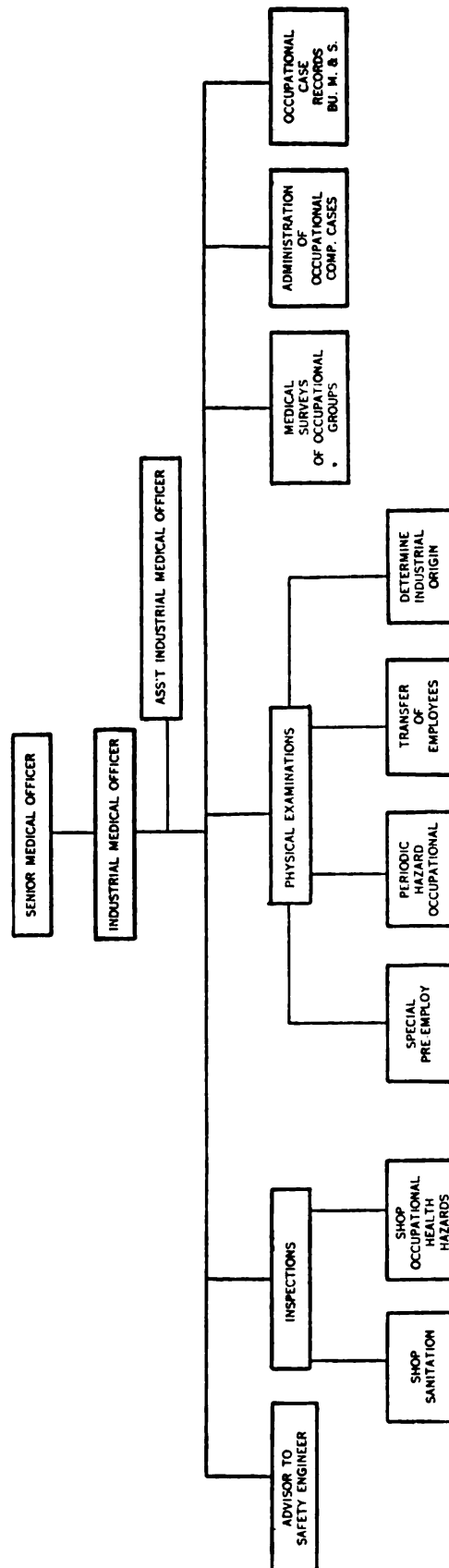


FIGURE 36.—Medical department organization for administration of industrial hygiene in a navy yard.

2. *Occupational health control*.—This lies within the sphere of both the safety engineer and the industrial medical officer.

THE INDUSTRIAL HYGIENE SURVEY.—This survey is a combined medical and engineering task.

1. *The medical survey*.—This consists of a complete study of all exposed personnel under the supervision of the industrial medical officer.

2. *The engineering survey*.—This falls under the direction of the safety engineer. It embraces a complete study of the environmental conditions. The safety engineer formulates the control program of the health hazard on the basis of the data obtained in the survey.

THE REPORTING OF INDUSTRIAL ACCIDENTS AND ILLNESS.—An important advance in accident prevention by the Navy was initiated on July 1, 1940, when the Secretary directed that a report of each accident and illness, both *lost-time* and *no lost-time* occurring among civil personnel of navy shore establishments be made to the Bureau of Medicine and Surgery, Navy Department. The report of each accident is submitted on a form, known as Form F-C. This presents the diagnosis and essential details as to cause of the injury. Punch cards are made up from these records for mechanical tabulating and indexing through a sorting machine. This provides facilities for the statistical analysis of these accidents and diseases.

OCCUPATIONAL HEALTH HAZARDS IN NAVY YARDS

DUST DISEASES.—(a) *Silicosis*.—All navy yards have iron, steel, and brass foundries and the dust-control problem is a major concern.

Due to the fact that silica (silicon dioxide) dust is not particularly irritating or obnoxious and the long period necessary for silicosis to develop, there is a tendency to an indifference toward this dust which must be overcome to accomplish permanent dust control. The medical control of silicosis consists of roentgen examination of the chest before employment and an annual roentgenogram of every man exposed to the higher silica dust concentrations, such as sandblasters and shakeout men.

(b) *Asbestosis*.—Asbestos dust is inhaled by workers in the manufacture of asbestos covers for flanges, valves, etc. Medical control consists of taking a roentgenogram of the lungs annually. The material is moistened, exhaust ventilation is installed over the work area, and a respirator is worn.

DISEASES DUE TO LEAD AND LEAD COMPOUNDS.—Lead poisoning has become comparatively infrequent in recent years due to changes in materials and methods and to improved measures of control.

DISEASES DUE TO VOLATILE ORGANIC SOLVENTS.—All spray lacquer personnel are subject to semiannual physical examinations.

DISEASES DUE TO ROENTGEN AND RADIUM HAZARDS.—These rays are utilized for the detection of flaws in castings, etc. Complete blood counts of all technicians are conducted quarterly and physical preemployment examinations are prescribed.

DISEASES DUE TO WELDING.—In naval welding much work is done in confined spaces. The chief hazards are *nitrous fume* poisoning and actinic ophthalmia from ultraviolet radiation of the welding arc. Nitrous fume poisoning is comparatively rare but a very serious condition. These injuries would be further reduced if control measures provided were properly utilized.

ANNUAL EXAMINATION OF CERTAIN EMPLOYEES

Crane operators, enginemen (hoisting and portable) and locomotive engineers are given annual physical examinations, with special emphasis on blood pressure, hearing, and vision. If corrective measures are impracticable the worker is retired or transferred to some suitable type of employment.

LOSS OF TIME

INDUSTRIAL VERSUS NONINDUSTRIAL DISABILITIES

In a limited study conducted by the American College of Surgeons a few years ago, it was found that the loss of time from nonindustrial types of illness was about 15 times that of industrial connected types. At the Washington Navy Yard in 1938, the time lost from nonindustrial disability was 37 times that lost from industrial causes. Much of this nonindustrial illness is preventable and can be greatly reduced by an annual physical examination of all employees. This would require a large increase of medical staffs but it would be a profitable investment.

STATISTICS

HEALTH OF THE NAVY

The statistics (annual rates per 1,000) appearing in this summary were compiled from data contained in monthly reports of communicable diseases received in the Bureau for the months of July, August, and September, 1940.

ENTIRE NAVY

Year	All diseases	Injuries and poisonings	All causes	Communicable diseases		Venereal diseases
				A	B	
1935-----	373	53	426	12	92	75
1936-----	336	67	404	24	88	49
1937-----	377	61	439	20	101	59
1938-----	332	57	389	7	72	81
1939-----	325	52	377	3	59	93
1940-----	386	50	436	16	104	82

FORCES ASHORE

1935-----	426	57	484	14	127	43
1936-----	416	69	486	29	120	38
1937-----	508	64	571	31	168	37
1938-----	342	52	394	10	92	39
1939-----	330	50	380	2	78	48
1940-----	367	50	417	13	114	51

FORCES AFLOAT

1935-----	343	51	395	11	72	92
1936-----	291	66	357	21	70	55
1937-----	302	60	362	13	63	72
1938-----	327	60	387	5	60	105
1939-----	322	54	376	4	49	117
1940-----	403	50	453	19	96	108

Common infectious diseases of the respiratory type.—There were 8,059 admissions for these diseases reported from the entire Navy for the third quarter of the year 1940—2,796 from the forces afloat, 2,558 from shore stations in the United States, and 2,705 from foreign shore stations. Catarrhal fever was responsible for 3,930 of the admissions.

Ships and shore stations reporting the largest number of cases were as follows:

Ship or station	July	August	September	Total
Naval Training Station, Norfolk, Va.....	352	147	144	643
Naval Training Station, Great Lakes, Ill.....	61	107	106	274
Marine Corps Base, San Diego, Calif.....	74	78	60	212
U. S. S. <i>California</i>	11	6	195	212
Marine Barracks, Quantico, Va.....	61	65	50	176
U. S. S. <i>Langley</i>	10	131	9	150
Fleet Air Base, Pearl Harbor, T. H.....	13	23	70	106
4th Marines, Shanghai, China.....	40	39	22	101
Naval Training Station, San Diego, Calif.....	33	33	31	97
Naval Air Station, Pensacola, Fla.....	36	34	25	95
U. S. S. <i>Enterprise</i>	32	19	41	92
Naval Training Station, Newport, R. I.....	39	34	16	89
Naval Air Station, Coco Solo, C. Z.....	8	54	16	78
U. S. S. <i>Medusa</i>	48	7	10	65
U. S. S. <i>West Virginia</i>	14	22	29	65
Submarine Base, Coco Solo, C. Z.....	19	33	6	58
U. S. S. <i>Portland</i>	5	21	27	53
U. S. S. <i>Augusta</i>	14	26	12	52
U. S. S. <i>Maryland</i>	12	22	18	52

Tonsillitis, acute.—U. S. S. *Minneapolis*.—Seventy-three cases of acute tonsillitis, representing more than 10 percent of the ship's complement, occurred among the enlisted personnel, between September 3 and 8, inclusive. Prior to the outbreak no case of sore throat or tonsillitis had been treated since August 5. The outbreak was explosive in type, the occurrence of cases by daily intervals, being 25, 31, 12, 3, 0, and 2. It is apparent that the number of cases declined abruptly after the third day and no new admissions occurred after the sixth day.

Only two men were hospitalized. One presented tremendous tonsils which, with the edema present, precluded swallowing and he received fluids and chemotherapy by venoclysis. The other developed a peritonsillar abscess.

The most prominent symptoms were pain in the throat and generalized aching. The typical case showed large cryptic tonsils studded with inflamed follicles or covered with a pseudomembrane. There was little cervical adenopathy. Temperatures ranged from 99.6° to 104° F.

The responsible organism was identified by culture as a nonhemolytic (gamma) type of streptococcus, and since 3 of the first 5 patients admitted showed this organism in smears from the tonsillar fossae all of the cases were given sulfanilamide. The drug was administered in a dosage of 15 grains every 4 hours with sodium bicarbonate 15 grains.

The therapeutic effect was apparent in nearly every case in 24 hours. In addition, each patient was given a hot saline gargle every 4 hours and aspirin as needed to relieve the general malaise. The commonest mild toxic reactions were headache and dizziness. Five patients developed cutaneous lesions, 4 of which resembled measles and could be distinguished from that affliction only by the absence of other associated signs. One patient developed severe urticaria after receiving 4.6 grams. This subsided rapidly when the drug was discontinued. Clinical results from the use of sulfanilamide were satisfactory.

Sixty-six of the 73 patients had not had tonsillectomies but only 6 gave a history of previous attacks.

Recovery was prompt in the great majority of cases and the average number of sick days was 6. Whether credit should be given to sulfanilamide, to the patients' resistance, or to the low virulence of the offending organism is not known.

The source of the outbreak remains obscure. The fact that it was explosive in type suggested a common source aboard ship, particularly since most of the patients had been on duty over the week-end and had remained on board. It is believed that a carrier can be ruled out because no case of tonsillitis or pharyngitis had been observed for approximately a month previously. The general mess was suspected, but cultures taken from mess gear at random in the scullery produced no growth. All food handlers and soda-fountain personnel were inspected and found to be free of any symptoms. Scuttle-butts were not considered to be the mode of transmission, because no mess attendants or chief petty officers became ill. Bacteriological examination of water in the ship's tanks was negative for pathogenic organisms.

Influenza.—U. S. S. *Langley*.—A total of 109 admissions for influenza between August 8–19, inclusive, was reported by the U. S. S. *Langley*. Sick days per case averaged 3.51. This epidemic was concurrent with a rather widespread epidemic of the disease among civilians in the Manila-Cavite area.

The most prominent symptoms were headache and pain in the muscles, particularly those of the lumbar group. A considerable number showed gastrointestinal symptoms, and cough and sore throat were fairly common complaints. All of the cases revealed mild inflammation of the upper respiratory tract but no case showed any unusual chest findings. Five cases ran a temperature of 105 or slightly over, while 26 cases showed a temperature of 104 or above. White blood counts tended toward a leukopenia.

One patient developed an otitis media, the only complication noted.

Other infectious diseases.—Cases of measles, German measles, and mumps were reported for July, August, and September as follows:

Ship or station	Measles	German measles	Mumps
U. S. S. <i>Augusta</i>	1	0	0
U. S. S. <i>Arizona</i>	0	10	1
Battle Force, Fleet Air Detachment.....	1	0	1
U. S. S. <i>Boise</i>	9	0	3
U. S. S. <i>California</i>	13	0	0
U. S. S. <i>Canopus</i>	0	0	2
U. S. S. <i>Chester</i>	1	0	2
U. S. S. <i>Chicago</i>	0	11	0
Submarine Base, Coco Solo, C. Z.....	0	0	1
U. S. S. <i>Colorado</i>	0	0	36
U. S. S. <i>Detroit</i>	0	32	0
U. S. S. <i>Enterprise</i>	0	2	0
U. S. S. <i>Holland</i>	0	2	1
U. S. S. <i>Indianapolis</i>	0	1	1
U. S. S. <i>Maryland</i>	6	5	16
U. S. S. <i>Minnesota</i>	0	1	0
U. S. S. <i>Mississippi</i>	21	1	13
U. S. S. <i>Nashville</i>	0	0	3
U. S. S. <i>Nevada</i>	5	0	1
U. S. S. <i>Pennsylvania</i>	4	0	0
U. S. S. <i>Philadelphia</i>	1	1	0
U. S. S. <i>Saratoga</i>	2	7	1
U. S. S. <i>Savannah</i>	0	1	1
U. S. S. <i>Tennessee</i>	2	17	0
U. S. S. <i>Trinity</i>	0	2	1
U. S. S. <i>West Virginia</i>	0	0	1
U. S. S. <i>Yorktown</i>	0	2	8
Naval Hospital, Chelsea, Mass.....	2	0	0
Submarine Base, New London, Conn.....	1	0	0
Naval Hospital, Philadelphia, Pa.....	0	0	1
Marine Barracks, Quantico, Va.....	1	1	0
Naval Air Station, Norfolk, Va.....	1	0	4
Naval Training Station, Norfolk, Va.....	0	18	18
Naval Hospital, Norfolk, Va.....	0	0	1
Navy Yard, Washington, D. C.....	0	0	1
Naval Air Station, Pensacola, Fla.....	6	0	0
Naval Hospital, Great Lakes, Ill.....	2	0	1
Naval Training Station, Great Lakes, Ill.....	12	0	10
Reserve Air Base, Long Beach, Calif.....	0	0	1
Marine Corps Base, San Diego, Calif.....	3	0	2
Naval Air Station, San Diego, Calif.....	7	3	0
Naval Training Station, San Diego, Calif.....	1	35	1
Receiving Ship, San Diego, Calif.....	5	0	3
Hospital Corps School, San Diego, Calif.....	3	3	0
Naval Air Station, San Pedro, Calif. (Terminal Island).....	1	0	0
Naval Hospital, Mare Island, Calif.....	0	2	1
Navy Yard, Mare Island, Calif.....	3	0	1
Naval Torpedo Station, Keyport, Wash.....	0	0	9
Puget Sound Naval Hospital, Bremerton, Wash.....	3	0	0
Puget Sound Navy Yard, Bremerton, Wash.....	0	1	0
Fleet Air Base, Pearl Harbor, T. H.....	0	0	1
Naval Hospital, Pearl Harbor, T. H.....	0	1	0

Ten cases of chickenpox were reported for the quarter, 6 from forces ashore and 4 from forces afloat.

Single cases of scarlet fever were reported in July from the U. S. S. *Tulsa* and the Research Laboratory, Anacostia, D. C.

Three cases of moderately severe typhus fever were admitted to the Regimental Hospital, 4th Marines, Shanghai, China, 1 in July, 1 in August, and 1 in September, with 28, 25, and 22 sick days, respectively.

A torpedo man, third class, 24 years of age, with 3 years and 3 months' service, was admitted to the sick list with cerebrospinal fever, meningococcic, on board the U. S. S. *Paul Jones* on September 12, 1940. He was transferred to the U. S. S. *Augusta* and died on September 13.

An aviation machinist's mate, second class, 27 years of age, with 9 years and 5 months' service, was admitted to the sick list with cerebrospinal fever, meningococcic, at the Naval Air Station, Norfolk, Va. on September 9, 1940. He was returned to duty on November 5.

A sergeant, Marine Corps, 37 years of age, with 15 years and 8 months' service, was admitted to the sick list at the Marine Corps Base, San Diego, Calif. on August 21 and transferred to the Naval Hospital, San Diego, Calif. on August 22 with meningitis, cerebral. The patient died on September 8, 1940.

A sergeant, Marine Corps, 32 years of age, with 12 years and 9 months' service, was admitted to the sick list at the Naval Training Station, Norfolk, Va. on July 6, 1940, with meningitis, cerebrospinal, acute. He was returned to duty after 31 days on the sick list.

The Naval Academy, Annapolis, Md. reported a case of meningitis, cerebrospinal, acute, in the case of a midshipman, first class, 22 years of age. He was admitted to the sick list on September 17 and discharged October 19.

A seaman, first class, 29 years of age, with 3 years and 11 months' service, was admitted to the sick list with typhoid fever at the Receiving Station, Norfolk, Va. on July 7, and discharged to duty on September 24.

The U. S. S. *Tutuila* reported a case of typhoid fever in the case of a seaman, first class, 21 years of age, with 2 years and 2 months' service. He was admitted to the sick list on September 11 and discharged to duty on October 17.

One case of paratyphoid fever "A" and 1 case of paratyphoid fever "B" were admitted in September on board the U. S. S. *Tutuila*.

Questionnaires for the cases of typhoid fever and paratyphoid fever have not been received in the Bureau, consequently no information is available concerning severity of case and prophylaxis history.

A case of poliomyelitis, anterior, acute, complicated by influenza, was admitted to the sick list on June 11, 1940 at the Regimental

Hospital, 4th Marines, Shanghai, China. The patient was transferred on August 5 to the U. S. S. *Chaumont* for transportation to the United States, and died on August 9.

Venereal disease.—The following table of statistical data indicates the frequency of occurrence of venereal diseases during July, August, and September, as compared with the previous 5-year median for the corresponding quarter:

Ship or station	Admission rate per 1,000	Median rate, 3rd quarter 1935-39
U. S. S. <i>Augusta</i>	322.09	226.07.
U. S. S. <i>Black Hawk</i>	263.93	296.06.
U. S. S. <i>Canopus</i>	410.71	219.85.
U. S. S. <i>Chaumont</i>	388.41	163.15.
U. S. S. <i>Chester</i>	127.72	68.11.
U. S. S. <i>Chicago</i>	152.72	55.81.
U. S. S. <i>Denebola</i>	147.40	Commissioned Jan. 16, 1940.
U. S. S. <i>Dixie</i>	174.12	Commissioned Apr. 25, 1940.
U. S. S. <i>Helena</i>	195.99	Commissioned Sept. 18, 1939.
U. S. S. <i>Honolulu</i>	175.77	Commissioned June 15, 1938.
U. S. S. <i>Langley</i>	424.46	65.27.
U. S. S. <i>Marblehead</i>	348.13	118.34.
U. S. S. <i>Nashville</i>	168.53	Commissioned June 6, 1938.
U. S. S. <i>Omaha</i>	495.64	113.04.
U. S. S. <i>Phoenix</i>	137.93	Commissioned Oct. 3, 1938.
U. S. S. <i>Quincy</i>	249.03	Commissioned June 9, 1936.
U. S. S. <i>Ranger</i>	152.56	91.08.
U. S. S. <i>San Francisco</i>	176.29	69.24.
U. S. S. <i>Saratoga</i>	163.42	86.35.
U. S. S. <i>St. Louis</i>	234.23	Commissioned May 19, 1939.
U. S. S. <i>Trenton</i>	250.50	94.03.
U. S. S. <i>Wasp</i>	190.63	Commissioned Apr. 25, 1940.
U. S. S. <i>Wichita</i>	310.59	Commissioned Feb. 16, 1939.
Naval Academy, Annapolis, Md. (other than midshipmen).	138.39	43.38.
Receiving Ship, New York, N. Y.---	149.49	94.02.
Receiving Station, Norfolk, Va.---	145.45	97.15.
Norfolk Navy Yard, Portsmouth, Va.---	174.96	83.18.
Naval Air Station, San Juan, P. R.---	251.97	Commissioned Sept. 18, 1939.
Submarine Base, Coco Solo, C. Z.---	186.23	126.65.
Navy Yard, Cavite, P. I.---	194.41	186.86.
4th Marines, Shanghai, China.---	253.16	143.32.
Marine Detachment, Tientsin, China.---	355.55	
Marine Barracks, Guam, M. I.---	212.12	61.07.
Naval Station, Guantanamo Bay, Cuba.---	127.73	75.71.

Gastro-enteritis.—Naval Air Station, Lakehurst, N. J.—Several cases of mild gastro-enteritis occurred in the Fifth Battalion, U. S. Marine Corps Reserve, from Washington, D. C., in the evening of August 31, the day before they were due to entrain for their return to Washington after completing their active-duty training. Out of approximately 500 men, 39 were ill and 10 were admitted to the sick bay overnight for observation and treatment. All but 1 case returned to duty the following morning. Mild nausea, abdominal

cramps, and diarrhea were the predominating symptoms. The Battalion returned to Washington before the cause of the outbreak could be determined.

Destroyer Division Ten.—During the period July 31 to August 10 there was an outbreak of gastro-enteritis aboard the ships of Destroyer Division Ten, 43 cases occurring on the U. S. S. *Cushing*, 41 on the U. S. S. *Preston*, 17 on the U. S. S. *Perkins*, and 3 on the U. S. S. *Smith*. The onset was abrupt in most cases and consisted of nausea, vomiting, mild abdominal cramps, and diarrhea, or any combination of these symptoms. No cases were acutely ill and only 8 cases requested to be excused from duty. No blood counts, bacteriological or other laboratory studies were done. In attempting to determine the cause of the outbreak the food, water, and sterilization of mess gear were considered, but the cause was not determined.

Food poisoning.—U. S. S. *Colorado*.—The Senior Medical Officer of the U. S. S. *Colorado*, under date of September 10, 1940, reported that 610 cases of food poisoning—5 officers and 605 enlisted men—occurred during the firing of short range battle practice on August 29. In addition, a number of observers from the U. S. S. *Mississippi* were sick from the same cause.

The first cases appeared at 11:00 a. m. The chief complaints were nausea and vomiting with violent stomach cramps, associated with pallor, a cold sweat, marked weakness, headache, rapid pulse, and evidence of acute fluid loss. A number of cases suffered from acute shock but rapidly recovered under treatment. Of the 610 cases, 478 were severe and 132 were mild.

The preceding meal, served at 9:45 a. m., consisted of egg-spread and corned-beef spread sandwiches. Specimens were sent to the U. S. S. *Relief* for examination within 12 hours, together with specimens of vomitus and diarrhea stools. The following report was received:

Egg specimen—Shows numerous gram positive cocci, many in chains.

Meat specimen—Occasional gram positive cocci.

The report of bacteriological studies on board the U. S. S. *Colorado* stated that both the egg and the meat showed a heavy contamination by the *Escherichia coli* and *Escherichia communior*.

The cause of the outbreak was believed to be the egg sandwich spread which was accidentally contaminated by enterotoxic staphylococcus, in spite of the fact that all galley rules and regulations regarding preparation and care of food were carefully carried out.

U. S. S. *Eagle* Boat #19.—Thirty-one cases of food poisoning occurred on board the U. S. S. *Eagle* #19 on July 31 from one to three hours after the 11:30 a. m. meal comprising fried fish, potato salad, and pineapple juice. The usual symptoms of nausea, vomiting, diarrhea, headache, and abdominal cramps were present. From a survey of the

patients, the causative foods were believed to be both the fish and the potato salad.

U. S. S. Wichita.—An epidemic of food poisoning affecting 56 persons occurred on board the *U. S. S. Wichita* on July 21 while moored in Rio de Janeiro, Brazil. The cause of the poisoning was attributed to ham served at the noon meal on general mess. Careful interrogation revealed that there was no blame for the evident contamination of one of the hams. The symptoms, diarrhea and vomiting with abdominal cramps, appeared about three hours after this meal. The cases were of a mild form and did not require admission to the sick list.

RECRUITS

In the sanitary report for July from the Marine Corps Base, San Diego, California, the medical officer reported that among 1,274 recruits examined there were found and charted 8,826 dental cavities, an average of 6.9 per man, as compared with 3.8 cavities per man for a six months' period in 1939. This would imply that recruits now being received present twice as much dental caries as recruits received a year ago.

MORBIDITY

Summary for the quarter ending Sept. 30, 1940

TABLE 1.—*Total, United States Navy*

Average strength.....	Forces afloat, 109,040		Forces ashore, 92,327		Entire Navy, 201,367	
	Admis- sions	Rate per 1,000	Admis- sions	Rate per 1,000	Admis- sions	Rate per 1,000
All causes.....	12, 340	452. 68	9, 617	416. 65	21, 957	436. 16
Diseases only.....	10, 973	402. 53	8, 470	366. 96	19, 443	386. 22
Injuries and poison- ings.....	1, 367	50. 15	1, 147	49. 69	2, 514	49. 94
Communicable dis- eases transmissible by oral and nasal discharges (Class VIII):						
(A).....	531	19. 48	293	12. 69	824	16. 37
(B).....	2, 623	96. 22	2, 632	114. 03	5, 255	104. 39
Venereal diseases.....	2, 939	107. 81	1, 177	50. 99	4, 116	81. 76

TABLE 2.—*Naval districts*

District	Average strength	Annual rates per 1,000			
		All causes	Diseases only	Respiratory diseases	Venereal diseases
1st Naval District.....	5, 256	347. 03	305. 17	94. 37	22. 83
3rd Naval District.....	4, 876	346. 18	301. 06	49. 22	63. 17
4th Naval District.....	3, 687	286. 41	251. 69	47. 74	34. 72
Activities on the Severn and Potomac Rivers.....	13, 984	354. 12	297. 48	80. 38	34. 04
5th Naval District.....	13, 381	550. 33	505. 79	218. 82	65. 47
6th Naval District.....	4, 851	386. 72	337. 25	56. 07	29. 68
7th Naval District.....	536	470. 15	388. 06	126. 87	29. 85
8th Naval District.....	4, 980	362. 25	306. 83	78. 71	27. 31
9th Naval District.....	5, 104	481. 19	423. 98	218. 65	17. 24
10th Naval District.....	534	501. 87	456. 93	82. 40	202. 25
11th Naval District.....	19, 983	334. 48	291. 45	83. 87	37. 03
12th Naval District.....	2, 567	280. 48	243. 08	46. 75	60. 77
13th Naval District.....	3, 016	343. 50	282. 49	53. 05	71. 62
14th Naval District.....	5, 345	347. 99	304. 58	136. 20	27. 69
15th Naval District.....	2, 167	695. 89	642. 36	275. 03	121. 83
16th Naval District.....	1, 135	489. 87	461. 67	84. 58	165. 64
Outlying stations.....	2, 878	890. 90	808. 90	204. 31	207. 09

DEATHS

During the quarter ending Sept. 30, 1940

Principal	Secondary or contributory	Total	Navy		Marine Corps		Midshipmen	Nurses
			Officers	Men	Officers	Men		
Average strength.....		201,367	13,039	153,207	1,917	30,378	2,356	470
<i>Disease</i>								
Abscess, perinephritic.....	Peritonitis, general, acute.....	1		1				
Adenocarcinoma, descending colon.....	Hemorrhage, intestinal.....	1		1				
Appendicitis, acute.....	Peritonitis, general, acute.....	2		2				
Bronchiectasis.....	Abscess, lung.....	1		1				
Carcinoma, histologic type unspecified, liver.....		1	1					
Cellulitis, buttock.....	Septicemia, organism unspecified.....	1		1				
Cellulitis, foot.....	Septicemia, staphylococcic.....	1		1				
Cellulitis, forearm and hand.....	do.....	1		1				
Cerebrospinal fever, meningococcic.....		1		1				
Coronary heart disease, arteriosclerotic.....		3	1	2				
Do.....	Pancreatitis, acute.....	1	1					
Edema, lung.....		1		1				
Endothelioma, pleura.....		1		1				
Glioma, histologic type unspecified, brain.....		1				1		
Hemorrhage, cerebral.....	Arteriosclerosis, general.....	1		1				
Do.....	Ulcer, stomach.....	1	1					
Hemorrhage, subdural.....		1		1				
Hypertensive heart disease.....		1		1				
Leukemia, acute, myelogenous.....		1		1				
Lymphosarcoma, mediastinum.....	Infarction, lung.....	1				1		
Melanoma, eye.....	Absence acquired, eye.....	1	1					
Meningitis, cerebral.....	Abscess, parotid.....	1				1		
Nephritis, acute.....		1		1				
Nephritis, chronic.....	Hypertension, arterial.....	1	1					
Do.....	Pneumonia, broncho.....	1		1				

DEATHS—Continued

Principal	Secondary or contributory	Total	Navy		Marine Corps		Mid-shipmen	Nurses
			Officers	Men	Officers	Men		
Pneumonia, broncho.....	Granulocytopenia, malignant.	1		1				
Pneumonia, lobar.....	Septicemia, staphylococci.	1		1				
Poliomyelitis, anterior, acute.		1				1		
Thrombosis, cerebral.....	Arteriosclerosis, general.	1		1				
Thrombosis, coronary, artery.		3		3				
Thrombosis, coronary, artery.	Arteriosclerosis, general.	1		1				
Thrombosis, superior mesentery.	Abscess, appendiceal.	1		1				
Tuberculosis, pulmonary, acute general miliary.		1		1				
Tuberculosis, pulmonary, chronic, active.		4		4				
Do.....	Tuberculosis, larynx and intestines.	1		1				
Ulcer, stomach.....	Fistula, duodenum.	1		1				
Valvular heart disease, aortic insufficiency.		1				1		
Total diseases.....		45	6	34	0	5	0	0
<i>Injuries and poisonings</i>								
Avulsion, leg.....	Gas bacillus infection, clostridium welchii, arm and leg.	1		1				
Crush, chest.....		1		1				
Decapitation.....		1	1					
Drowning.....		9	1	8				
Fracture, compound, ribs.	Hemorrhage, traumatic, pulmonary.	1		1				
Fracture, compound, skull.		8		7		1		
Fracture, simple, ribs....	Wound, punctured, liver and lung.	1		1				
Fracture, simple, skull....	Hemorrhage, traumatic, intracranial.	1		1				
Do.....	Intracranial injury.	1				1		
Fracture, simple, vertebra, cervical.	Intraspinal injury.	1				1		
Fracture, simple, vertebra, thoracic.	do.....	1		1				
Fracture, simple, vertebrae, cervical and thoracic.	do.....	1		1				
Heat stroke.....		1		1				
Injuries, multiple, extreme.		24	5	16		3		
Intracranial injury.....		5	2	1		2		
Do.....	Pneumonia, broncho.....	1	1					
Strangulation, respiratory.		2		2				
Wound, gunshot, head....		4	1	2	1			
Do.....	Psychosis, unclassified.	1		1				
Wound, incised, neck.....	Dementia praecox.	1		1				
Wound, lacerated, thigh..	Gas bacillus infection, organism unknown, leg.	1		1				
Poisoning, therapeutic, acute, sulfanilamide, gonococcus infection, urethra.	Gonococcus infection, urethra.	1		1				
Total for injuries and poisonings.		68	11	48	1	8	0	0
Grand total.....		113	17	82	1	13	0	0
Annual death rate per 1,000:								
All causes.....		2.24	5.22	2.14	2.09	1.71		
Diseases only.....		.89	1.84	.89		.66		
Drowning.....		.18	.31	.21				
Poisonings.....		.02		.03				
Other injuries.....		1.15	3.07	1.02	2.09	1.05		

MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

Statistics for the quarter ending September 30, 1940

The following statistics were taken from sanitary reports submitted by naval training stations.

July, August, and September 1940	Naval training station			
	Norfolk, Va.	Newport, R. I.	Great Lakes, Ill.	San Diego, Calif.
Recruits received during the period.....	4, 715	3, 068	4, 209	3, 761
Recruits appearing before board of medical survey...	72	32	28	0
Recruits recommended for discharge from the Service.....	55	17	28	20
Recruits discharged by reason of medical survey...	45	(1)	22	(1)
Recruits held over pending further observation...	40	20	(1)	0
Recruits transferred to the hospital for treatment, operation, or further observation for conditions existing prior to enlistment.....	0	15	(1)	55

¹ Not reported.

The following table was prepared from reports of medical surveys in which disabilities or disease causing the survey were noted existing prior to enlistment. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office.

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Abdominal adhesions.....	4	Constitutional psychopathic state, inadequate personality.....	4
Abscess, periapical.....	1	Constitutional psychopathic state, sexual psychopathy.....	4
Absence, acquired, kidney.....	1	Contracture.....	1
Absence, acquired, teeth.....	4	Cryptorchidism.....	3
Adhesions, intestinal.....	1	Curvature, spine.....	1
Amblyopia.....	3	Cyst.....	2
Arthritis, chronic.....	2	Deafness, bilateral.....	3
Asthma.....	3	Deafness, unilateral.....	4
Astigmatism, mixed.....	1	Defective physical development.....	1
Astigmatism, compound, hyperopic.....	1	Deformity, acquired.....	13
Bronchitis, chronic.....	1	Deformity, congenital.....	7
Cardiac arrhythmia, auricular, extra systole.....	1	Dementia praecox.....	6
Cardiac arrhythmia, paroxysmal tachycardia.....	2	Diabetes mellitus.....	1
Cardiac arrhythmia, premature contractions.....	1	Dislocation, articular cartilage.....	1
Caries, teeth.....	9	Dislocation, chronic.....	7
Choroiditis.....	1	Dysinsulinism.....	1
Cicatrix, skin.....	5	Effort syndrome.....	1
Color blindness.....	4	Encephalitis, chronic.....	1
Constitutional psychopathic inferiority, without psychosis.....	5	Enuresis.....	39
Constitutional psychopathic state, emotional instability.....	8	Epilepsy.....	27
		Erb's syphilitic spinal paralysis.....	1
		Flat foot.....	21

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Fracture, simple, thoracic vertebra.....	1	Psychoneurosis, anxiety neurosis.....	1
Genu valgum.....	1	Psychoneurosis, hysteria.....	5
Gonococcus infection, urethra.....	4	Psychoneurosis, neurasthenia.....	5
Hallux valgus.....	1	Psychoneurosis, situational.....	1
Hammertoe.....	1	Psychoneurosis, traumatic.....	2
Hay fever.....	2	Psychosis, traumatic.....	1
Headache.....	3	Pyelitis, chronic.....	1
Heart disease.....	1	Rheumatic fever.....	3
Hernia, inguinal, indirect.....	15	Rupture, traumatic.....	1
Hernia, ventral.....	1	Sinusitis, maxillary.....	1
Hydronephrosis.....	1	Somnambulism.....	11
Hypertension, arterial.....	1	Strain, muscle.....	2
Hypertrophy.....	1	Sprain, joint, sacro-iliac.....	1
Hypothyroidism.....	1	Stuttering.....	1
Hypotension, arterial.....	1	Syncope.....	2
Insufficiency, ocular muscle.....	1	Synovitis.....	2
Laryngitis, chronic.....	1	Syphilis.....	3
Loose body in joint.....	2	Syphilis, sero-positive only.....	8
Malocclusion, teeth.....	1	Tachycardia.....	4
Metatarsalgia.....	1	Tuberculosis, pulmonary, chronic, active, moderately advanced.....	2
Myocarditis, chronic.....	1	Tuberculosis, pulmonary, chronic, arrested, moderately advanced.....	1
Myopia.....	6	Ulcer, duodenum.....	8
Myositis, chronic.....	3	Ulcer, stomach.....	1
Myotonia, congenital.....	1	Union of fracture, faulty.....	2
Narcolepsy.....	1	Valvular heart disease, aortic and mitral.....	1
Nephritis, chronic.....	5	Valvular heart disease, aortic stenosis.....	1
Neuritis, optic.....	1	Valvular heart disease, mitral insufficiency.....	3
Neuritis, sciatic.....	1	Valvular heart disease, mitral stenosis.....	3
Nostalgia.....	1	Varicocele.....	1
Obstruction, intestinal, from external causes.....	1	Varicose veins.....	2
Osgood-Schlatter disease.....	1		
Osteochondritis dissecans.....	2		
Osteomyelitis.....	1		
Otitis media, chronic.....	34		
Otosclerosis.....	1		
Pansinusitis.....	1		
Paradentosis.....	3		
Pes cavus.....	1		
Prostatitis, chronic, nonvenereal.....	1	Total.....	394

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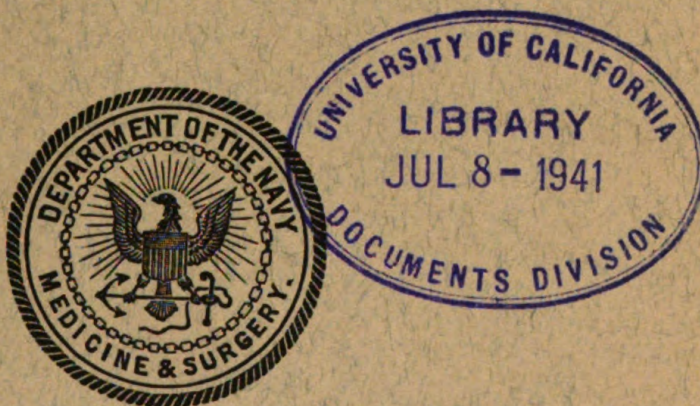
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THE MISSION OF THE MEDICAL DEPARTMENT OF THE NAVY



TO KEEP AS MANY MEN AT AS MANY GUNS AS
MANY DAYS AS POSSIBLE



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NAVY DEPARTMENT,
Washington, March 20, 1907

This UNITED STATES NAVAL MEDICAL BULLETIN is published by direction of the Department for the timely information of the Medical and Hospital Corps of the Navy.

TRUMAN H. NEWBERRY,
Acting Secretary.

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PREFACE

THE UNITED STATES NAVAL MEDICAL BULLETIN was first issued in April 1907 as a means for supplying medical officers of the United States Navy with information regarding the advances which are continually being made in the medical sciences, and as a medium for the publication of accounts of special researches, observations, or experiences of individual medical officers.

It is the aim of the Bureau of Medicine and Surgery to furnish in each issue special articles relating to naval medicine, descriptions of suggested devices, clinical notes on interesting cases, editorial comment on current medical literature of special professional interest to Medical Department personnel, and reports from various sources, notes, and comments on topics of professional interest.

The Bureau extends an invitation to all medical and dental officers to prepare and forward, with a view to publication, contributions on subjects of professional interest.

In order that each service contributor may receive due credit for his efforts in preparing matter for the BULLETIN of distinct originality and special merit, the Surgeon General of the Navy will send a letter of appreciation to authors of papers of outstanding merit.

The Bureau does not necessarily undertake to endorse views or opinions which may be expressed in the pages of this publication.

ROSS T. MCINTIRE,
Surgeon General, United States Navy.

NOTICE TO CONTRIBUTORS

Contributions to the **BULLETIN** should be typewritten, double spaced, on plain paper and should have wide margins. Fasteners which will not tear the paper when removed should be used. Nothing should be written in the manuscript which is not intended for publication. For example, addresses, dates, etc., not a part of the article, require deletion by the editor. The **BULLETIN** endeavors to follow a uniform style in heading and captions, and the editor can be spared much time and trouble, and unnecessary changes in manuscript can be obviated if authors will follow in these particulars the practice of recent issues.

The greatest accuracy and fullness should be employed in all citations, as it has some times been necessary to decline articles otherwise desirable because it was impossible for the editor to understand or verify references, quotations, etc. The frequency of gross errors in orthography in many contributions is conclusive evidence that authors often fail to read over their manuscripts after they have been typewritten.

Contributions must be received at least 3 months prior to the date of the issue for which they are intended.

The editor is not responsible for the safe return of manuscripts and pictures. All materials supplied for illustrations, if not original, should be accompanied by reference to the source and a statement as to whether or not reproduction has been authorized.

The **BULLETIN** intends to print only original articles, translations, in whole or in part, reviews, and reports and notices of Government or departmental activities, official announcements, etc. All original contributions are accepted on the assumption that they have not appeared previously and are not to be reprinted elsewhere without an understanding to that effect and that editorial privilege is granted to this Bureau in preparing all material submitted for publication.

LOUIS H. RODDIS, *Editor,*
Commander, Medical Corps, United States Navy.
HILTON W. ROSE, *Assistant Editor,*
Lieutenant Commander, Medical Corps,
United States Naval Reserve.

U. S. NAVAL MEDICAL BULLETIN

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SPECIAL ARTICLES

BRUCELLOSIS

UNDULANT FEVER

By Commander Walter M. Simpson, Medical Corps, United States Naval Reserve¹

INTRODUCTION

Brucellosis (undulant fever, Malta fever, Mediterranean fever, goat fever, contagious or infectious abortion of cattle, Bang's disease, Texas fever, Rio Grande fever, et cetera) is now known to be a common and widely distributed disease of animals and man. Our studies of this disease, begun in 1928, resulted in the discovery of 90 cases in and about Dayton during a period of 18 months. In 1930, the author expressed the belief that brucellosis would soon become recognized as a major public health problem. That this opinion was justified has been abundantly substantiated by developments of the past decade.

The first cases of Malta fever to be recognized in this country occurred among men recently returned from the Tropics (Musser and Sailer, 1899). Craig (1905) not only detected many cases among men in the Army who had served in the Philippine Islands, but also established the diagnosis in a nurse who had never been out of the country and who had had no contact with patients with Malta fever. Craig suggested at this time that many patients with atypical typhoidlike fever might be suffering from Malta fever. A few cases of the disease were reported from goat-raising areas in Texas, New Mexico, and Arizona by Gentry and Ferenbaugh (1911) and by Yount and Looney (1913). Then the disease apparently faded from medical consciousness for a decade, when Lake and Watkins (1922) published their startling report of an epidemic of Malta fever in Phoenix, Arizona, which they traced to the infection of raw goat's milk. Up to this time physicians naturally associated brucellosis only with a goat source. It was the monumental observation by Alice Evans (1918), that the organisms of Malta fever and contagious abortion

¹ Director, Kettering Institute for Medical Research, Miami Valley Hospital, Dayton, Ohio.

of cattle were for all practical purposes indistinguishable which led to the discovery of the widespread occurrence of *Brucella* infection of bovine and porcine origin.

Following the first report by Keefer (1924) of a case of brucellosis due to the abortus variety of the organism, a rapid succession of reports of cases of bovine or porcine origin appeared from South Africa, Canada, Germany, Sweden, Norway, Denmark, Italy, Great Britain, Switzerland, The Netherlands, France, Puerto Rico, and New Zealand. Subsequent reports leave little doubt that brucellosis is world-wide in its distribution and is most prevalent in those areas in which *Brucella* infection of cattle, hogs, and goats is widespread.

For 21 years the *Micrococcus melitensis* of Bruce and the *Bacillus abortus* of Bang had been regarded as separate, unrelated species, until Alice Evans' (1) observation that the two organisms are indistinguishable morphologically, biochemically, culturally, and by ordinary agglutination tests. The results of further studies led Evans to state that the two organisms probably possessed similar pathogenicity for human subjects. In 1927, Carpenter (2) recovered an organism indistinguishable from *Brucella abortus* from the blood of 10 human beings suffering from brucellosis; 5 pregnant heifers inoculated with these cultures promptly aborted. These observations of Evans and Carpenter have been confirmed by many workers throughout the world. It is now known that the organism which produces contagious abortion in cattle and other domestic animals is capable of producing in human beings a disease clinically and bacteriologically similar to the Mediterranean type of Malta fever.

Since Bruce had described the organism as a coccus and Bang had termed it a bacillus, it became apparent that the melitensis-abortus group of organisms should be reclassified. The proposal by Meyer and Shaw (3) that the organisms should be designated by the generic name *Brucella* has met with universal approval. Consistent with this proposal, the name brucellosis has been generally adopted as the single designation for the disease produced in animals and man by the *Brucella*. The organism usually associated with goat infection is termed *Brucella melitensis*, while the organism of infectious abortion of cattle is called *Brucella abortus*. Traum (4) described a third related organism associated with the infection in swine, which has been designated *Brucella suis*.

Organisms which have been designated as caprine (melitensis) or porcine (suis) have been recovered from cow's milk. The antigenic differences are probably best explained on the basis of host adaptation. It seems probable that *Brucella abortus* is the primitive strain from which other strains have developed as the result of passage through various animal hosts.

The importance of brucellosis as a rapidly developing public health problem is vividly indicated by recent experiences with the disease in the United States. Prior to 1926, the sporadic cases which were encountered were regarded as clinical curiosities. Most of these were related to endemic foci of goat infection in Texas, New Mexico, and Arizona. During the years 1927 to 1930, the recorded cases numbered 217, 649, 1301, and 1385, respectively. During 1929, cases of brucellosis were encountered in every State of the Union. In 1940, there were 3,358 cases officially reported to the United States Public Health Service by State health departments (5). It is undoubtedly true that the number of cases actually occurring is much larger than that reported. Hardy (6) reported the occurrence of 1,669 cases in one State (Iowa) from 1925 to 1935. It has become more and more apparent that this rapid increase in the incidence of the disease has been due chiefly to infection of human beings with the organisms of contagious abortion of cattle and hogs. American physicians soon realized that these observations provided an explanation for their failure to arrive at a confirmed diagnosis in many cases of typhoid-like and malaria-like diseases.

Among urban populations, the disease appears to be transmitted through the raw milk of cattle infected with *Brucella abortus*. Of the 248 patients with brucellosis studied personally by the writer, the ingestion of raw milk or unpasteurized dairy products containing the organism of contagious abortion of cattle was demonstrated to be the source of infection in the great majority of instances; these findings are corroborated by the investigations of Carpenter, King, Orr, Huddleson, Farbar, Mathews, Sensenich, Giordano, Ey, and others. Hardy expresses the belief that direct contact with infected cattle and hogs has been responsible for a great number of cases of brucellosis occurring in Iowa. Hardy's investigations of 991 cases of brucellosis occurring in Iowa, led him to the conclusion that the abortus and suis species of the organism are about equally responsible for the brucellosis morbidity in that State. Hardy (7) has demonstrated by animal experiments that the skin may act as the portal of entry of the organism. Morales-Otero (8), of Puerto Rico, has reproduced the disease in human volunteers by inoculations through abraded skin. It is apparent, therefore, that there are two important sources of infection for man, that is, the ingestion of raw milk or unpasteurized dairy products containing *Brucella*, or direct contact with infected fresh animal tissues or discharge. *There is no definite evidence of man-to-man transmission of the disease.*

CLINICAL MANIFESTATIONS IN MAN

Because brucellosis presents many symptoms and signs common to typhoid fever, malaria, tuberculosis, and influenza, it is frequently confused with these diseases. Many physicians have arrived at a tardy diagnosis of brucellosis only after repeated negative Widal reactions, the failure to demonstrate the malarial plasmodium, and the inability to elicit physical signs or x-ray evidence of tuberculosis. Less often, the disease has been confused with acute rheumatic fever, subacute bacterial endocarditis, bronchitis, pyelitis, appendicitis, cholecystitis, or tularemia.

SYMPTOMATOLOGY

As knowledge of the clinical manifestations of brucellosis has advanced, particularly during the past decade, it has become more and more apparent that the older textbook descriptions of the symptomatology of the disease were based largely on experiences with brucellosis of goat origin. The classical clinical picture of a disease characterized by an undulatory, remittent, or intermittent fever, drenching sweats, chills, headache, backache, muscular and joint pains, weakness, loss of weight, possibly a palpable spleen, an infrequent skin eruption, leucopenia with lymphocytosis, and secondary anemia, pertains chiefly to the severe acute forms of the disease. In a high proportion of cases, the disease pursues a relatively mild, prolonged course, extending over many months or years. The early descriptions by Hughes (9) and Craig (10) included many such cases. Alice Evans and others have more recently directed particular attention to the common chronic ambulatory form of brucellosis, in which the patient, whose spirits are already depressed by continued ill health, is often given further discouragement when a diagnosis of neurasthenia is made. Many neurasthenics, whose chief complaints were exhaustion, insomnia, irritability, and a variety of aches and pains, have been found to be victims of chronic brucellosis.

Since the symptomatology of the acute and the chronic forms of brucellosis varies so greatly, these two manifestations will be considered separately.

ACUTE BRUCELLOSIS

The incubation period has been found to vary from 5 days to longer than 1 month. In accidental laboratory infections the incubation period has varied from 10 to 20 days. The prodrome is not unlike that of any general infection, with a gradual onset, although in occasional cases the disease is initiated with a sharp chill and a rapid

elevation of temperature to 103°–105° F. (39.4°–40.6° C.). A sense of tiredness and weakness, loss of appetite, constipation, headache, and backache are common early symptoms. Usually, the patient becomes gradually aware of an afternoon or evening rise in temperature, associated with chills or chilly sensations, nocturnal perspiration, and weakness. The patient often feels quite well during the morning hours, particularly in the early stages of the infection. As the temperature rises, during the afternoon or evening, the symptoms gradually return and increase in severity. The nocturnal exacerbations of fever occasionally reach great heights (106°–107° F.; 41.1°–41.7° C.). There is often a remarkable disparity between the subjective sense of fever and the height of the fever as registered by the clinical thermometer; in many instances the patient does not complain of fever, nor does he present a febrile appearance, but the physician finds, to his surprise, a fever of 101°–103° F. (38.3°–39.4° C.). As the fever abates, chills and sweating occur. If defervescence is rapid, the perspiration is more likely to be of a drenching character. In such cases, the sweats, which literally saturate the night clothing and bedding, are one of the most impressive features of the disease. The perspiration often has a peculiar sweetish, fetid odor. The chills may be severe enough to be regarded as true rigors in about one-third of the acute cases (11, 12, 13, 14). Many patients experience only mild chilly sensations, while in about one-fourth of cases chilliness is absent.

Arthralgia and muscular pains are prominent features of the acute form of the disease in approximately one-half of the cases. The joint pains may be more pronounced during the onset or they may persist throughout the course of illness. The myalgia may be accompanied by a feeling of stiffness not unlike the muscular soreness which follows vigorous exercise. Hydrarthrosis and transient periarticular swelling have been observed occasionally. Permanent impairment of the joints usually does not occur. Suppurative osteomyelitis as a complication of brucellosis has been described with increasing frequency during the past few years (15, 16, 17, 18, 19, 20, 21, 22, 23). In most of the reported cases the vertebrae, particularly in the lumbar region, were attacked. In some instances other bones, such as the humerus, femur, skull, and ribs, were affected. While the osteomyelitis sometimes occurred relatively early in the course of the disease, in most cases it was a late complication, occasionally occurring after apparent recovery from acute brucellosis. Feldman and Olson (24, 25) found similar examples of *Brucella* spondylitis in 24 hogs, an average occurrence of 1 in every 6,000 swine slaughtered.

Marked restlessness and insomnia usually accompany the nocturnal febrile exacerbations. Delirium occurs in some cases in which the fever reaches great heights. Regional *Brucella* localizations in the

brain, spinal cord, or meninges may occur during the acute phase of the disease and produce symptoms and signs of encephalitis, myelitis, or meningitis; such complications are, however, more commonly observed as delayed manifestations of brucellosis. According to Roger and Poursines (26), whose recent classical monograph has aroused great interest in the frequency of central nervous system invasion by *Brucella*, the meningeal involvement predominates, and the development of encephalitis or myelitis, or both, is usually secondary to the meningitis. Because of this predilection for meningeal localization, Roger and Poursines have termed this form of the disease *meningo-neurobrucellosis*. The involvement of the central nervous system may produce the first and only symptoms of the disease. The symptomatology varies greatly, depending upon the extent of meningeal invasion and the presence of additional complications involving the brain, spinal cord, or peripheral nerves. In a patient with brucellosis, the development of such symptoms and signs as severe headache, vertigo, diplopia, nuchal rigidity, aphasia, psychic disturbances and various forms of paralysis which are often evanescent, calls for examination of the cerebrospinal fluid. Characteristically, the spinal fluid will be under increased pressure and will show pleocytosis, increase of albumin, and a decrease of globulin and sugar. Since the ultimate diagnosis during life depends upon the isolation and identification of *Brucella* from the spinal fluid, a particularly diligent effort should be made to recover the organism by culture and by guinea pig inoculation. De Jong (27) has recorded 11 verified cases of *Brucella meningitis* or *meningoencephalitis* in which the organism was recovered from the spinal fluid by culture or by guinea pig inoculation.

The matutinal remissions or intermissions and the nocturnal exacerbations of fever may last from one week to many months. The inadequate name, undulant fever, refers to recurring relapses of fever following afebrile intermissions. Such febrile relapses are the exception rather than the rule; most patients experience but one febrile period, lasting from a few days to several months, and finally reaching the normal level by lysis.

The essential gastro-intestinal complaints are anorexia and constipation. The degree of constipation appears often to parallel the severity of the infection. Diarrhea is of rare occurrence. Nausea and vomiting may occur in the more severe cases. Abdominal pain is a not infrequent feature of the disease during its early manifestations. Among 125 cases studied by Hardy, abdominal pain occurred in 40; in 10 it was the major complaint. Simpson found abdominal pain to be the chief complaint in 16 of 142 patients with brucellosis. The occurrence of abdominal pain has often led to appendectomy in patients with unsuspected brucellosis, the correct diagnosis having

been made only after the persistence of febrile symptoms stimulated a further search for the cause. As in typhoid fever, the gall bladder may become a focus of *Brucella* infection. *Brucella* has been recovered from the excised gall bladder or from the bile following duodenal drainage in patients with symptoms of cholecystitis (28, 29, 30).

Symptoms referable to the respiratory tract may be an outstanding feature of the disease in certain instances. Cough, associated with mucoid or mucopurulent sputum production, is not infrequent during the first few weeks of illness, and may persist for months. Recent reports (31, 32, 33, 34, 35) provide convincing evidence that pulmonary lesions of brucellosis are of frequent occurrence and are often detectable by roentgenographic examination, even in the absence of distinctive physical signs of pneumonia. The lesions most often encountered on x-ray examination are peribronchial infiltrations, hilar infiltrations, and scattered discrete or confluent patchy pneumonic areas. The pulmonary manifestations of brucellosis should be regarded as hematogenous lobular pneumonia rather than true bronchopneumonia. Without doubt, pulmonary lesions occur much more commonly than is generally realized. Serial roentgenographic studies will often provide an explanation for vague respiratory symptoms.

The most serious cardiovascular complication has been the occasional occurrence of vegetative endocarditis. Smith and Curtis (36) found reports of nine cases of *Brucella* endocarditis confirmed by post-mortem examination, to which they added a similar case. In most instances the vegetations occurred on mitral or aortic valves previously damaged by rheumatic fever.

The name commonly applied to brucellosis of cattle, infectious abortion, is derived from the well-known predilection of the causative organism for the genital tract. There is strong evidence that the same regional localization sometimes occurs in human beings. Painful swelling of the testes has been described frequently. Acute epididymitis, orchitis, prostatitis, and seminal vesiculitis may be early manifestations of the disease. Simpson (12) recovered *Brucella abortus* from a draining sinus tract which extended from the globus major of the epididymis through the scrotal wall.

There appears to be little doubt that brucellosis is at least an occasional cause of abortion in women who live on farms where they have direct contact with infected animals, or in women who consume raw milk or unpasteurized dairy products. There are reports (37, 38, 39, 40) of human abortion in which the history and serologic findings provide strong circumstantial evidence of the etiologic role of *Brucella*. In a study of 565 cases of brucellosis, Calder (41) found a history of one or more miscarriages in 32 percent of the married women; a history

of one miscarriage followed by sterility was common; a few women reported as many as 5 or 6 abortions. More direct evidence has been provided by Carpenter and Boak (42) who recovered *Brucella abortus* from the tissues of a human fetus which was aborted at the end of the fourth month of gestation. Kristensen (43) isolated the abortus variety of the organism from the exudate which covered the uterine site of the placenta of a 7-month fetus. Frei (44) isolated *Brucella* from the vaginal discharge of a woman who had aborted 10 days previously.

Loss of weight is an almost constant feature of the acute form of the disease. The greatest loss, often from 10 to 50 pounds, occurs in those patients who experience high fever, drenching sweats, and great prostration.

A transient cutaneous eruption, usually papular, macular, or maculopapular, is a relatively infrequent finding. The skin lesions may simulate the roseola of typhoid fever.

CHRONIC BRUCELLOSIS

Many physicians feel that the symptoms and signs of acute brucellosis are often sufficiently characteristic to justify such a provisional diagnosis on the basis of clinical findings. In dealing with chronic brucellosis, however, the physician is often faced with a problem which will tax his diagnostic acumen to the utmost. No disease, not excepting syphilis and tuberculosis, is more protean in its manifestations.

Quite naturally, a certain amount of wholesome skepticism has arisen in the minds of some physicians regarding any wide prevalence of chronic brucellosis. Such a feeling of doubt is quite defensible, since one is justified in questioning the validity of some of the diagnostic tests upon which such a diagnosis is often based.

The common and unfortunate employment of the name "undulant fever" has served only to add further difficulties in the recognition of cases of chronic brucellosis. A significant temperature curve, physical signs of disease, and positive agglutination tests and skin tests may be entirely lacking throughout a long period of chronic illness. The recent studies by Evans (45, 46, 47), Poston (48), Angle (49), Scoville (50), Thames (51), Calder (41), Hamman and Wainwright (52), Harris (53), and Cameron and Wells (54) leave little doubt that a protracted, relatively mild form of brucellosis is widely prevalent and constitutes a major cause of chronic ill health. Only a small proportion of patients with chronic brucellosis, probably less than 10 percent, have experienced a previous acute febrile illness, compatible with a diagnosis of acute brucellosis. In many cases the patient is not entirely incapacitated for work, but complains

chiefly of weakness and exhaustion, with or without mild fever. Since the commonly employed diagnostic tests are frequently negative in such cases, and since even the most conscientious physician may not find physical abnormalities to account for the patient's complaints, the almost inevitable diagnosis of neurasthenia or psychoneurosis is often made.

Surveys (55, 56) conducted in widely separated parts of the United States during recent years reveal that an ambulatory, partially disabling, chronic form of brucellosis is a widespread cause of prolonged ill health. Angle, Algie, Baumgartner, and Lunsford (57) found that 9 percent of 7,122 school children gave positive reactions to the intradermal test; 79.3 percent of the positive reactors consumed raw milk; a high proportion of the children had complaints consistent with the ambulatory type of brucellosis. Gould and Huddleson (55) observed positive reactions in 845 of 8,124 persons (10.3 percent) tested intradermally with brucellergin. Most of these individuals were residents of an infirmary for homeless indigents and of a mental hospital where unpasteurized milk was consumed and where the supply of dairy products was obtained partly from a herd known to be infected with *Brucella*. Of the 845 reactors to the skin test only 111 (13.1 percent) showed a positive agglutination reaction. The unreliability of the agglutination test in detecting chronic brucellosis has been noted by many other workers.

A recitation of all of the symptoms which have been ascribed to chronic brucellosis would serve only to heighten the confusion which as yet surrounds this baffling phase of the disease. In general, it may be stated that the three cardinal features of most cases of chronic brucellosis are weakness, low-grade fever and a lack of objective physical findings. McGinty and Gambrell (58) have listed over 150 different manifestations of chronic brucellosis. Mild degrees of fever may be present for many weeks or months; there may be several months of complete freedom from fever; sudden febrile exacerbations may occur, accompanied by an accentuation of the prevailing symptoms, or by the development of evidence of new regional symptoms affecting the respiratory, cardiovascular, genitourinary, gastro-intestinal, skeletal or nervous systems. Pneumonia, endocarditis, orchitis, epididymitis, prostatitis, oophoritis, cholecystitis, hydrarthrosis, arthritis, spondylitis, osteomyelitis, ocular complications, or meningoencephalitis may be associated with the acute form of the disease, but much more commonly appear several months, or even years, after the often indefinite onset of the chronic form of brucellosis. In some instances such delayed evidences of regional *Brucella* localization may appear long after apparent recovery from the acute manifestations of the disease. All students of chronic brucellosis have emphasized the

almost universal prominence of symptoms which relate to the central nervous system. In addition to the occasional acute invasion of the meninges, brain, and spinal cord by *Brucella*, there is evidence (59) that the endoantigen of *Brucella* organisms circulating in the blood has a toxic action upon the central nervous system. These observations led Evans (45) to state: "These facts challenge the right of a physician to make a diagnosis of neurasthenia—a diagnosis regarded as dishonorable by the patient, and also by his family, his employer, and his friends—without considering, among other possibilities, the possibility of chronic brucellosis."

Chronic brucellosis should be suspected in all cases of so-called fever of unknown origin. There are many reports of the isolation of *Brucella* from the blood, urine, bile, or from extirpated tissues in patients who have experienced unexplained, long-continued, low-grade fever for years. Hamman and Wainwright (52) reexamined 36 such patients; an accurate diagnosis was finally made on 10 of them; 3 were found to have brucellosis.

In contrast to acute brucellosis, it is of particular significance that chronic brucellosis has been recognized in relatively few areas in the United States. The discovery of a large number of cases in these centers has been largely the result of a determined effort on the part of a few investigators to learn of the incidence of the disease in their localities. It seems quite apparent that the vast majority of cases remain unrecognized, since the evidence at hand indicates that chronic brucellosis is widely prevalent in rural communities and in cities and towns in which raw milk is consumed.

DIAGNOSIS

If brucellosis is given consideration in the differential diagnosis of all cases of febrile illness, especially in those in which the diagnostic criteria for typhoid fever, tuberculosis, influenza, malaria, chronic bronchitis, pyelitis, rheumatic fever, or bacterial endocarditis are not convincing, the disease will be recognized with much greater frequency. This is true in cases of vague, mild febrile disease as well as in those in which the clinical manifestations of brucellosis are more clearly defined. In such cases it should become an established practice to submit 4 or 5 cc. of the patient's blood, collected exactly as for the Wassermann test, to a laboratory equipped with the proper *Brucella* antigens for the agglutination test. The rapid macroscopic agglutination method of Huddleson is a simple and reliable procedure.

Since it is exceedingly hazardous to base a diagnosis of brucellosis solely on clinical grounds, recourse must be had to laboratory diagnostic tests. These procedures include (*a*) primary isolation of the causal organism by cultural methods from blood, spinal fluid, secre-

tions, excretions, or excised tissues; (b) indirect recovery of *Brucella* by culture after animal inoculation; (c) the agglutination test; (d) the intradermal test; and (e) the opsonocytophagic reaction.

The only method by which the diagnosis of brucellosis may be completely established is by the cultivation and identification of the organism. While cultural technics have improved greatly during the past few years, with a corresponding increase in the number of reported instances of recovery of the organism, the undertaking is often beset with difficulties and requires skill and, above all, patience. The *melitensis* and *suis* varieties of *Brucella* ordinarily grow readily under aerobic conditions, while the much more commonly encountered *abortus* variety requires an atmosphere containing 10 percent carbon dioxide. The procedure used by Poston (48) with notable success is as follows: 15 cc. of blood are obtained from each patient by venipuncture and placed in a small flask containing 4 cc. of sterile 2.5 percent sodium citrate solution. Four flasks containing 100 cc. of liver infusion broth of pH 6.8 are each inoculated with 2 cc. of the citrated blood. The flasks are incubated at 37° C., two in the room atmosphere and two in an atmosphere containing 10 percent CO₂. After 4 days' incubation, daily smears of the broth cultures are made and stained by Gram's method. If no organisms are seen in the smears after 10 days' incubation, 5 cc. of the original culture are transplanted to 100 cc. of liver infusion broth every 3 days for 2 weeks. Original cultures and transplants are incubated for 3 weeks before they are reported as negative.

The guinea pig is the most suitable laboratory animal for inoculation. Poston inoculates three guinea pigs with blood from each patient; two are injected intraperitoneally with 2 cc. each of citrated blood; one is inoculated in the groin with 1 cc. of citrated blood. The animals are observed daily. Beginning 2 months after inoculation, tests for specific agglutinins and for cutaneous reaction to Huddleson's brucellergin are made at intervals of a few days. When both tests become positive the animals are killed. Animals which remain negative to the agglutination test and to the skin test are killed 4½ months after inoculation. Liver infusion broth is planted with the guinea pig's blood and with pieces of organs and is subjected to the cultural procedures previously described. The cultures may then be differentiated into *abortus*, *suis*, or *melitensis* varieties by the agglutinin-absorption technic, the bacteriostatic action of dyes, glucose utilization, and hydrogen sulphide metabolism (60, 61, 62).

The most commonly used and the most reliable indicator of *Brucella* infection, in the absence of positive cultures, is the agglutination test. This is particularly true in cases of acute brucellosis, in which a high serum agglutinin titer will be found in a great majority of

cases. Agglutinins may appear as early as the fifth day of illness, but ordinarily are not found until the second week after the onset. In some instances, specific agglutinins may not appear for several weeks. One important source of difficulty in interpreting the results of agglutination tests is the fact that agglutinins may be persistently absent (in about 6 percent of patients) or may be present in low titer in persons from whom *Brucella* has been cultivated. Another source of error in interpreting the agglutination reaction is the fact that the titer may remain at a high level for months or years after recovery. Then, too, some individuals exposed to the infection may develop agglutinins without notable illness. Furthermore, the level of the agglutinin titer may fluctuate widely on repeated testing. *These considerations call for the exercise of keen judgment in interpreting the results of the agglutination test. A person suffering from some disease other than brucellosis may have a positive agglutination test merely as the result of a previous symptomatic or asymptomatic Brucella infection.*

In the past, diagnostic significance has usually been attributed to titers of 1:80 or above. The choice of such an arbitrary diagnostic titer is not justified in the light of recent studies. In those cases in which the clinical manifestations suggest brucellosis, the absence of agglutinins or their presence in titers of 1:10 to 1:40 should stimulate further bacteriologic and serologic studies.

The difficulties which attend the interpretation of agglutination tests in cases of acute brucellosis are greatly multiplied in cases of chronic brucellosis. While the great majority of patients with the acute form of the disease reveal a positive agglutination test in high titer, a high proportion of patients with chronic brucellosis give repeated negative agglutination reactions or positive tests in low titer. In a group of 28 cases of chronic brucellosis studied by Evans (63), 46 percent gave a negative agglutination reaction.

The occasional cross agglutination of *Brucella* and *Bacterium tularensis* should be borne in mind. In cases of tularemia the relatively higher titer with the *B. tularensis* antigen and the usually typical history leaves little doubt as to the interpretation of the serologic findings. If the *Brucella* and *B. tularensis* titers are the same, or nearly so, agglutinin absorption tests will distinguish between them.

While the agglutination test is undoubtedly of great value, its limitations must be recognized. Otherwise, errors will be made in two directions: First, the correct diagnosis of brucellosis may not be made because too much reliance is placed in a negative test; or second, an incorrect diagnosis of brucellosis may be made in a person who has a residual agglutinin titer from a previous invasion by Brucella, but who is suffering from some other disease when the test is made.

The *intradermal* test is used to determine cutaneous hypersensitivity to specific *Brucella* antigen. A positive allergic skin reaction is generally accepted as evidence of past or present *Brucella* infection. While the great majority of patients from whose blood *Brucella* has been recovered show a positive skin test, the test has yielded negative results in rare instances in which *Brucella* infection was proved by culture.

The chief source of error in interpreting the significance of a positive skin test lies in the fact that the test is frequently positive in exposed individuals with no history of an illness compatible with brucellosis. Furthermore, the hypersensitivity, once acquired after symptomatic or subclinical infection, usually persists for many years. *Therefore, it must be emphasized that a positive skin test does not mean that the symptoms from which the patient is suffering at the time of a positive skin test are necessarily due to brucellosis.* Students of this disease are only too familiar with instances in which a diagnosis of brucellosis was made only on the basis of a positive skin test and in which further developments revealed the presence of some such disease as active tuberculosis, Hodgkins' disease, leukemia, typhoid fever, malaria, or subacute streptococcic endocarditis. The intradermal test is regarded by Evans as a less accurate indicator of present infection than the agglutination test because the allergic state usually develops later than agglutinins and because it is generally retained for longer periods after recovery. Gould and Huddleson (55), on the other hand, regard the skin test as the most sensitive diagnostic test for brucellosis; these investigators express the belief that if the skin test is negative, brucellosis may usually be ruled out.

A variety of antigens has been used for skin testing. The two agents most commonly employed are (a) a heat-killed suspension of *Brucella* in physiologic saline solution (vaccine) and (b) a suspensoid of nucleoprotein isolated from *Brucella* by chemical separation, known as Brucellergin (Huddleson). If commercially available vaccines are used for skin testing, the usual procedure is to dilute the vaccine in a proportion of 1 part vaccine to 9 parts sterile physiologic solution of sodium chloride and to inject 0.1 cc. of the diluted suspension intracutaneously in the ventral surface of the forearm. It is important to select properly standardized vaccines from a reliable source. Variations in the manner of preparation, potency, and dosage have led to a lack of uniformity in the production and interpretation of cutaneous reactions. Brucellergin has the advantage of being a standardized preparation, especially designed for skin testing. The brucellergin test is also performed by injecting 0.1 cc. into the skin of the forearm. A positive reaction by either method is characterized by the development of a circumscribed erythematous, edematous, indurated area at the site of injection. In a positive test,

the area of local reaction averages about three-fourths of an inch in diameter, but may vary from $\frac{1}{2}$ inch to 3 or more inches. The reaction usually reaches its greatest intensity in 24 to 48 hours; ordinarily it is best to observe the results of the test 48 hours after injection. The presence of mild, transient erythema, without edema and induration, is of no significance. In frankly positive cases the induration usually persists for several days. In hypersensitive persons a positive test may be accompanied by a mild, or in some instances a severe, systemic reaction. In such cases the lymphatic channels above the site of inoculation may become red, thickened and painful, and the regional axillary lymph nodes may become enlarged and tender. An exacerbation of symptoms may follow the development of a positive skin test. Focal necrosis at the site of inoculation occurs in a small number of cases.

There is evidence (64, 65) that the injection of heat-killed *Brucella* for the intradermal test stimulates the production of agglutinins. Evans (47) found that the intracutaneous injection of brucellergin in 12 volunteers caused the development of opsonins in 7 and agglutinins in 5; in one instance the agglutinins rose from zero to a titer of 1:320. *Hence it is advisable to collect blood specimens for serologic tests before the intradermal test is performed.*

The inadequacies of the agglutination test and the intradermal test, particularly in distinguishing between present and past *Brucella* infection, led Huddleson, Johnson, and Hamann (66) to reintroduce the *opsonocytophagic reaction*, a modification of the Leishman-Veitch technic for determining the phagocytic activity of the blood in the presence of serum opsonins and homologous leucocytes. The opsonocytophagic test is employed in conjunction with the intradermal test or the agglutination test, or both, to determine the immunity status of an individual giving positive tests by either or both methods. The test is performed by mixing 0.1 cc. of the patient's citrated blood with 0.1 cc. of a saline suspension of living *Brucella* which have been grown for 48 hours on liver infusion agar. The suspension should contain at least 6 billion organisms per cc. of physiologic saline solution (pH 7.0). The mixture is then incubated at 37° C. (98.6° F.) for 30 minutes, after which a small amount of the sedimented cells is removed with a capillary pipette. A smear is then made from a large drop of the cell suspension, dried rapidly and stained with Hasting's stain or Bordet-Gengou's carbol toluidin blue. The number of bacteria in 25 polymorphonuclear neutrophilic leucocytes is determined and classified according to the number of bacteria per polymorphonuclear leucocyte; the absence of phagocytosis indicates a negative result; 1 to 20 phagocytized bacteria indicates slight, 21 to 40 moderate, and 41 or more, marked phagocytosis.

According to Huddleson's interpretation, the opsonocytophagic power of the blood is low during the active infective phase of the disease and becomes marked after recovery. On this basis, it is considered that individuals have developed immunity to *Brucella* if 60 percent or more of the polymorphonuclear leucocytes show marked phagocytosis. If as many as 40 percent of the leucocytes show moderate to marked phagocytosis the patient may be infected and has not yet developed any immunity, or he may be uninfected.

While theoretical considerations lend support to the contentions of Huddleson and his associates as to the value of the opsonocytophagic test, it still lacks confirmation. The studies by Calder (41), and Keller, Pharris, and Gaub (67, 68) appear to provide some support to Huddleson's thesis. On the other hand, Evans (63) regards the opsonocytophagic test as the least reliable of the diagnostic tests in cases of chronic brucellosis; she found strongly positive (immune) reactions in 4 cases from which *Brucella* were cultivated, and weak or moderate reactions in recovered cases. Morales-Otero and Gonzalez (69) tested over 200 individuals (cattle handlers, milkers, and laboratory workers exposed to *Brucella* infection) with a purified *Brucella* skin-test antigen, agglutination tests, complement fixation tests, and opsonocytophagic tests. They found no correlation between cutaneous allergy to *Brucella* and the opsonocytophagic reaction. Fifteen cases that were positive to the opsonocytophagic reaction gave a negative cutaneous reaction, while 18 showing a positive cutaneous reaction were completely negative to the opsonocytophagic reaction. Lee Foshay (70, 71) and the writer, working independently, have found that a significant proportion of patients yield aberrant and unexpected results in relation to their immunity status when the opsonocytophagic test is employed in conjunction with cultural methods, agglutination tests and skin tests. High phagocytic titers (immune reactions) occur in some patients with severe and uninterrupted brucellosis, proved by cultures. Certain recovered patients, asymptomatic for months or years, exhibit marked fluctuations from month to month, running the entire gamut from high to low phagocytosis, or sometimes none at all. *Until more extensive studies have been made on culturally proved cases of brucellosis the results of the opsonocytophagic test should be interpreted with caution and with reservations.*

Hematocytologic studies indicate that leucopenia, with an increase in lymphocytes at the expense of polymorphonuclear leucocytes, occurs in about two-thirds of cases; in the remaining third, moderate leucocytosis or normal leucocyte counts occur. Mild to moderate degrees of anemia are usually present. The erythrocyte sedimentation rate frequently exhibits a moderate increase.

A lowered basal metabolic rate, vascular hypotension, gastric hypo-

chlorhydria, or achlorhydria are commonly observed in patients with chronic brucellosis.

Just as the diagnosis of brucellosis may be missed by too great reliance upon any one or all of the diagnostic tests, so also is there danger that the diagnosis may be made much too freely by unjustified reliance on a positive skin test. This hazard pertains particularly to the diagnosis of chronic brucellosis.

PROGNOSIS

Fatal outcome is rare, having occurred in about 2 percent of reported cases. One hundred and seven deaths from brucellosis were officially recorded in the United States during 1936. The importance of the disease is not to be judged by the low mortality rate. The prolonged course and the resulting chronic ill health in a high proportion of cases make the outlook much more serious than the death rate would indicate. Death is usually the result of overwhelming acute infection, terminating fatally during the first few weeks of illness, or it follows a relapse at any stage of the disease due to regional localizations of *Brucella* in such structures as the meninges, brain, heart valves or lungs.

TREATMENT

The most important consideration in the control of brucellosis is *prophylaxis*. The widespread distribution of the infection among cattle renders it difficult to control the infection at its source. Many cows have Bang's disease and eliminate the organisms in large numbers in the milk and vaginal discharges without manifesting symptoms of the disease (abortion, mastitis, sterility, and lessened milk yield).

Since 1934, the United States Department of Agriculture has been engaged in a laudable campaign directed toward eradication of brucellosis in cattle. After 55 months of diligent effort, ending January 31, 1939, more than a million and a half cattle (approximately 5.5 percent of the number tested) were found to give positive tests for brucellosis; the infected cattle were condemned and the farmers and dairymen received indemnities to compensate for the loss of the cattle. Unfortunately, no similar campaign has yet been inaugurated to control the disease in hogs and goats.

There is but one logical method for preventing the transmission of milk-borne infection to human beings, and that is by pasteurization. Brucellosis is only one of the formidable list of diseases transmitted to man through the use of raw milk and other unpasteurized dairy products. Murray, McNutt, and Purwin (72) Boak and Carpenter (73), and Zwick and Wedeman (74) have demonstrated that complete pasteurization (143°–145° F. (61.6°–62.7° C.) for 30 minutes) will destroy *Brucella*. The need for strict supervision of the pasteurization process

is apparent. For the protection of the health of those persons whose occupations bring them in direct contact with infected animal tissues we must rely upon education and the institution of precautionary measures.

For those persons who live on farms, or in small communities where pasteurization is not yet practiced, home pasteurization may be carried out by placing the milk in an aluminum vessel and heating it to 155° F. (68.3° C.), stirring constantly, then immediately setting the vessel in cold water and continuing the stirring until cool.

The general management of the patient with brucellosis will be largely determined by the prevailing symptoms and signs. General therapeutic considerations will vary greatly in individual patients. The therapeutic program for patients with acute brucellosis is quite different from that employed for the chronic form of the disease.

In severe cases of acute brucellosis, the general supplementary management is essentially that employed for any acute febrile disease, characterized by fever, chills, and sweats. If the sweating is of a drenching character special provisions should be made for the protection of the mattress and for changes of bed linen and gowns. If the fever exceeds 104° F. (40° C.) tepid sponge baths are indicated; *antipyretic drugs should be avoided*. The patient should remain constantly in bed during the febrile phase and for at least one week after the temperature has returned to the normal level. Skilled nursing care is an essential requirement in the severe cases. The same precautions should be taken as regards the disinfection of urine and feces as in cases of typhoid fever. As the fever abates, a liberal diet of high caloric value is required, particularly for patients who lose considerable weight. The patient should be cautioned against resuming even moderate activity until there has been an appreciable gain in strength. Two or three additional weeks of rest will often save the patient from several months of partial disability.

In cases of chronic brucellosis, the general management must be strictly individualized. Since most of these patients are ambulatory and are at least attempting to perform some work, often at great effort, the program should be directed towards a maximum amount of rest. This may entail a regulation of the hours of work in order to avoid excessive fatigue. Since most of these patients suffer from the symptoms ordinarily ascribed to neurasthenia, a generous amount of practical psychotherapy, largely directed towards reassurance, is a fundamental requirement. The members of the patient's family should be fully informed as to the nature of the illness in order to enlist their sympathetic understanding and to avoid their stigmatizing the patient as neurotic or as a malingerer. The common symptoms of headache, backache, joint and muscle pains, insomnia,

anorexia, constipation, and anemia usually yield to appropriate symptomatic measures.

Specific therapy.—While there is considerable evidence that the employment of various types of serum therapy and vaccine therapy has greatly improved the outlook for most patients suffering from acute or chronic brucellosis, it is extremely difficult to evaluate the effectiveness of any form of specific therapy in a disease characterized by natural remissions and by an extremely variable symptomatology. The reported results of vaccine therapy or serum therapy run the entire gamut from pessimism to hyperenthusiasm. More extensive controlled and systematic studies on a large number of patients, carried out over a period of many years, are necessary before definite statements can be made. It would appear, however, that sufficient data have been accumulated to justify the continued and extended use of some of the specific agents.

Serum therapy.—Interest in serum therapy, which had waned following the earlier appearance of several unfavorable reports, has been revived by the development of a more potent anti-*Brucella* serum by Foshay (75, 76) and his associates at the University of Cincinnati. Detoxified *Brucella* antigens are employed for the development of the antisera in goats or horses. Several favorable experiences with the Foshay serum have been recorded by other workers (77, 78, 79, 80, 81). This type of antiserum therapy should be restricted to patients with acute or subacute brucellosis, preferably to those who have had the disease less than 8 months. The dosage recommended by Foshay is as follows: For adults suffering from moderately severe to severe manifestations of the disease, the average total dose is 60 cc., given by three daily intravenous or intramuscular injections of 20 cc. each or by two daily injections of 30 cc. each; in unusually severe infections, 90 to 120 cc. may be given in unit doses of 30 cc. during a period of 48 to 72 hours; for children, a total of 20 to 30 cc. may be given, either intramuscularly or subcutaneously, in daily doses of 10 cc. each.

Serum therapy is not indicated in cases of chronic brucellosis of more than 8 months' duration, unless sudden, severe exacerbations occur. Such abrupt relapses are usually the result of regional localizations involving the meninges, brain, spinal cord, heart valves, lungs, liver, spleen, and bone marrow. In such cases, the dosage of serum would be that recommended for unusually severe infections.

Scattered reports of the use of convalescent human serum, blood transfusions from recovered donors, or immuno-transfusions from donors who have received prior injections of heat-killed *Brucella*, have appeared (82, 83, 84, 85, 86). Even though the number of cases is small and the period of observations not sufficiently long for accurate

evaluation, the results were sufficiently gratifying to justify further trials, particularly as regards immuno-transfusions.

Vaccine therapy.—It seems probable that the earlier discrepant reports of the effectiveness of vaccine therapy had their basis in a lack of standardized methods for the preparation of the vaccines, both as regards the choice of suitable strains and the concentration of the vaccine. These difficulties appear to have been largely overcome in recent years by the development of better standards for the preparation of therapeutic vaccines.

Brucella melitensis (varieties *abortus* and *suis*) vaccine, N. N. R., has been widely employed and is available through the usual trade sources. This vaccine is a saline suspension of heat-killed or formalin-killed *Brucella abortus* and *suis* organisms in equal quantities. Vaccines prepared from the *melitensis* variety of the organism should be utilized only in the treatment of the relatively rare *Brucella melitensis* infections.

Experience has taught that no rigidly standardized scheme of dosage of vaccine is applicable to patients with brucellosis. Experience and good judgment are essential requisites in determining the proper dosage for each individual. The usual procedure with the commercially available vaccine is first to test for hypersensitiveness by injecting 0.05 cc. of a 1:10 dilution of the vaccine intracutaneously. If the patient does not experience an excessive local or systemic reaction within the next 48 hours, an initial therapeutic dose of 0.25 cc. is injected into the deep subcutaneous tissues, or preferably into the muscle. Local reactions are minimized by intramuscular injections. If no untoward reaction follows the first injection of 0.25 cc., a second dose of 0.25 cc. is given 3 days later. The dosage is then increased in increments of 0.25 cc., at intervals of 3 days, until a dosage of 1 cc. is reached. Ordinarily, two injections of 0.5 cc. and two of 0.75 cc. are given before the 1 cc. dosage is attained. Five to eight injections of 1 cc. each may then be given at 3-day intervals.

If the patient is highly sensitized, it is wise to begin with intramuscular doses of 0.1 cc., or, in rare instances of extreme sensitization, with doses of 0.1 cc. of a 1:10 to 1:100 dilution of the vaccine, and gradually increase the dosage by 0.1 cc. increments until a dosage of 1 cc. is reached. If, during the course of vaccine injections an unusually severe local or systemic reaction should occur it is desirable to reduce the next dose to one-half the amount which produced the severe reaction and then cautiously and gradually increase the succeeding doses.

A series of 4 to 6 or more sharp systemic, febrile reactions, usually accompanied by a transient exacerbation of symptoms, is the goal of the treatment. Hence, only extreme local or general reactions

should be avoided. Elevations of temperature to 103°–105° F. (39.4°–40.6° C.) are not uncommon within 4 to 8 hours after the injection of vaccine. Such systemic responses may occur following the first injection of a small quantity of vaccine or may not occur until relatively large doses are given. In chronic brucellosis larger doses of the vaccine may be required; if no reaction is provoked after five or six 1 cc. injections, the dosage may be gradually increased by 0.5 cc. increments to 2 or 3 cc.

While some patients who have obtained an apparently satisfactory response to vaccine therapy have had little or no thermal reaction, the most prompt and lasting results have occurred in those who have experienced several such reactions.

Erythema and tenderness at the site of vaccination occur commonly for a day or two following injections. In about 5 percent of cases, a local hard tumefaction may persist for much longer periods. In a small proportion of such cases sterile abscesses or local areas of necrosis have developed.

Brucellin therapy.—Brucellin is a fraction of *Brucella* cells obtained by growing the organism in liver broth. The bacteria-free active agent is recovered from the liver broth filtrate. This preparation was devised by I. F. Huddleson (87, 88, 89) and may be procured at the Central *Brucella* Station, Michigan State College, East Lansing, Mich.

The dosage of Brucellin must also be adjusted to suit the requirements of individual patients. After the extent of sensitiveness has been determined by the intradermal injection of 0.1 cc. of Brucellin, the usual procedure in nonhypersensitive patients is to give repeated injections of 1 cc. at intervals of 3 days until the morning and evening temperatures between the intervals of injection tend to become subnormal. Here again, one object of this form of therapy is the production of a series of four or more febrile, systemic reactions. If the duration of illness is less than 10 weeks, the likelihood of recovery following four 1 cc. injections is greater than if the duration is longer than 10 weeks. Patients with long-continued chronic brucellosis require a larger number of injections and may require gradually increasing amounts up to 5 cc. before satisfactory reactions are produced. In highly sensitized persons, it is advisable to start with intramuscular doses of 0.1 cc. If there is no severe systemic reaction following this injection, each succeeding dose may ordinarily be doubled, until the larger dosage is attained.

In children, the initial dose of *Brucella* vaccine or Brucellin should not exceed 0.1 cc. and succeeding doses should be increased by not over 0.1 cc. increments. Considerable dilution of the vaccine is required for hypersensitive children.

A partially oxidized detoxified vaccine, devised by Foshay and O'Neil (90) has been used with apparent success (41). Much smaller doses are given subcutaneously at more frequent intervals. The few reports of results indicate the recovery rate equals that of other vaccines or Brucellin. Local or constitutional reactions do not occur with the oxidized vaccine. It has been recommended chiefly for the treatment of chronic brucellosis.

While it is difficult to evaluate the results of vaccine or Brucellin therapy, the experiences of many investigators indicate that about 60 percent of patients with brucellosis obtain apparently complete recovery after a satisfactory course of either agent. An additional 25 percent appear to obtain some benefit, while the remaining 15 percent are not improved.

The contraindications to vaccine or Brucellin therapy are heart disease, renal disease, arteriosclerosis, meningeal or cerebral localizations of *Brucella* or the acute fulminating (malignant) form of the disease.

Nonspecific protein therapy.—Injections of foreign protein substances, such as sterile skimmed milk, typhoid vaccine or typhoid-paratyphoid vaccine have been utilized (91, 92, 93) for the production of nonspecific shock reactions in the treatment of brucellosis. Erwin and Hunt (94, 95) reported good results in 20 patients with acute and subacute brucellosis following the intravenous injection of killed typhoid-paratyphoid organisms. The usual initial dose was 30 to 50 million killed organisms, with 2 to 6 additional injections, increasing the dosage by increments of 25 million organisms.

Chemotherapy.—Neoarsphenamine, mercurochrome, acriflavine, metaphen, thionin, methylene blue, methyl violet, gentian violet, and other chemical substances have been used in the treatment of brucellosis. In most instances the reports of the apparently successful use of these substances were based upon observations limited to small numbers of patients. The very length of the list argues against the specificity of any of them.

Sulfanilamide and related compounds have been heralded (96) as effective agents in the treatment of brucellosis since 1936. After the first wave of enthusiasm, usually based on short observations on relatively few patients, other reports of less favorable or entirely negative results have appeared. Blumgart and Gilligan (97) analyzed the results reported in the 31 papers which appeared between 1936 and 1939. Twenty-four of the reports were concerned with only 1 or 2 patients. Of the 74 cases treated with sulfanilamide or allied compounds, there were 68 apparent recoveries and 6 failures; 14 of the 68 patients (20 percent) exhibited relapse after apparent recovery. The daily dosage of sulfanilamide employed in most cases was 4 to 6 gm. (60 to 90 grains) during the period of fever, with

gradually diminishing dosages for 3 or 4 days after the fever abated. The administration of the drug was rarely continued for more than 12 days. Bynum (98) reported 6 cases of brucellosis unsuccessfully treated with large doses of sulfanilamide. Long and Bliss (99) report recurrence of infection in 4 of 5 patients whose immediate response to sulfanilamide therapy was apparently quite satisfactory; in 2 instances *Brucella* was recovered from the blood after sulfanilamide therapy was discontinued. The writer has had similarly disappointing experiences in several cases treated with large doses of sulfanilamide, controlled by determinations of the sulfanilamide concentration of the blood. *Until more extensive and extended studies are made on culturally proved cases, the value of sulfanilamide therapy in cases of brucellosis must be regarded as undetermined.* In this connection it might be well to recall the fact that a temporary remission is not synonymous with cure.

Artificial fever therapy.—The observation that recovery from brucellosis often followed the induction of fever by chemical or biological agents led Prickman and Popp (100) to investigate the possible usefulness of artificial fever induced by physical means in the management of brucellosis. Each of 3 patients was given 3 artificial fever treatments, each of 5 hours' duration, at a rectal temperature of 105°–106° F. (40.6°–41.1° C.); all were benefited by the treatment. Zeiter (101) described a similarly favorable experience. More recently, Prickman, Bennett, and Krusen (102) analyzed the results of treatment with physically induced hyperpyrexia in 21 cases of brucellosis; apparent cure resulted in 80.9 percent of the patients. The duration of the disease prior to artificial fever therapy varied from 10 days to 2½ years. The authors expressed the belief that fever therapy was most efficacious in the acute and subacute febrile stages of the disease. Simpson (103), who has had similar favorable results, reserves artificial fever therapy for those refractory patients who do not respond to vaccine therapy. Artificial fever therapy should be carried out only in properly equipped institutions by thoroughly qualified physicians and nurse-technicians.

Supplementary therapy.—Since some degree of anemia is a common finding in brucellosis, it should be combated with appropriate dietary, iron, or liver therapy. Repeated small transfusions (200 cc.) are of value in the management of the more severe grades of anemia. In the more chronic forms of the disease, in which severe anorexia is a prominent feature, vitamin deficiency is common. Large doses of vitamin B, with reinforcement of vitamin A, C, and D intake, has been effective in relieving this distressing symptom.

SUMMARY AND CONCLUSIONS

1. The author's prediction of a decade ago, greeted with some skepticism at that time, that brucellosis would become recognized as a public health problem of major proportions has been fulfilled. Of the 248 cases of brucellosis studied by the author, all but 52 occurred prior to the passage of a universal pasteurization ordinance in 1931. In each of the 52 cases occurring during the past 10 years the patient had consumed raw milk elsewhere prior to the onset of illness.

2. The symptomatology of the acute and the chronic forms of brucellosis varies greatly. The diagnostic criteria for acute brucellosis are usually not applicable to the chronic form of the disease. There is little doubt that the chronic ambulatory form of brucellosis is widely prevalent, is often confused with other diseases, and frequently is not recognized. Many "neurasthenics" and patients with so-called "fever of unknown etiology" have been found to be victims of chronic brucellosis. Less than 10 percent of patients with chronic brucellosis have experienced a previous acute febrile illness, compatible with a diagnosis of acute brucellosis.

3. The only diagnostic procedure by which the diagnosis of brucellosis may be established with certainty is the cultivation and identification of the organism. The agglutination test and skin test are of considerable value in the diagnosis of acute brucellosis, but these procedures are notoriously inadequate as diagnostic aids in cases of chronic brucellosis. A positive agglutination test, particularly of low titer, and a positive skin test do not indicate that the person is suffering from brucellosis at the time the tests are made. Both the agglutination test and the skin test will yield entirely negative results in an appreciable number of persons from whose blood *Brucella* may be recovered.

4. In our hands, the opsonocytophagic test has yielded a high proportion of inconsistent results. We have found this test to be of little or no value as a diagnostic procedure or as a guide to therapeutic response.

5. Since it is now well established that brucellosis is caused most frequently by the ingestion of raw milk containing *Brucella*, the most important consideration in the control of the disease is adequate, controlled pasteurization of all milk and dairy products.

6. *Brucella* vaccine therapy has produced favorable results in from 60 to 85 percent of patients with either acute or chronic brucellosis. Sulfanilamide and other sulfonamide drugs are apparently of little benefit. Artificial fever therapy has yielded favorable results, particularly in those refractory patients who did not respond to vaccine therapy.

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COMPRESSED AIR ILLNESS

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This paper brings up to date the review of the literature on compressed air illness which was published in this Bulletin in 1938 (1). The study will be presented under the following subject heads: Cause, symptoms, treatment, prophylaxis, and general unclassified bibliography. Also, as before, only compressed air illness will be considered and no articles dealing with related subjects will be reported. All literature reviewed and reported was published during the calendar years 1936 to 1940, inclusive. This work will of necessity appear disjointed and unfinished as it is merely a supplementary report and not in any sense intended to be a comprehensive study. No review or discussion of generally accepted theories is included. As a matter of fact, it is assumed that the reader either has a working knowledge of compressed air illness or that he reads this in conjunction with my original complete review.

CAUSE

The accepted theory of the cause of compressed air illness is that of gaseous emboli in the blood stream and tissues. Almost all writers since 1916 and many prior to that time have accepted this theory and few have even considered any other possibility. Shaw (2) dis-

cusses the cause with special reference to the formation, distribution and behavior of the nitrogen bubbles. He gives as an explanation of the delayed development of symptoms, the growth of the bubbles by accretion as a result of simple diffusion due to the difference in partial pressure in the tissues and in the small newly formed bubbles. Reminiscent of some of the older theories, we find Lipkovic (3) discussing the influence of the soil and of the amount of air inspired on the incidence of compressed air illness. His observations on caisson workers are sound for increased percentages of CO₂ (usually due to improper ventilation rather than gas seepage from the soil) have been shown by many authors to have a marked effect in increasing the incidence of compressed air illness.

It is well known by all who work in this field that certain facts relative to the development of compressed air illness are difficult to explain satisfactorily on the basis of the physical phenomenon of air embolism alone. One of these is the long period of time frequently noted between decompression and the development of symptoms; another is that after prolonged exposure over the calculated maximum saturation time it is impossible to decompress safely according to standard tables; and still another is the fact that a man may work for weeks without any trouble and then one day develop serious or fatal symptoms without any of the conditions of work, exposure, or decompression having been changed. The author (unpublished research) worked on the proposition that the discrepancies might be explained by blood chemical changes. An observation of value in the interpretation of these conflicting data was reported by Swindle (4) in 1937. He called attention to what appears to be a normal physiological phenomenon characterized by the intravascular agglutination of erythrocytes. This may be the incipient stage of intravascular coagulation or by breaking apart may release fragile emboli consisting of clumps of cells. The next year End (5) in discussing the cause of compressed air illness says: "It may be stated at this time that agglutination of erythrocytes appears to be the primary disturbance in compressed air illness and that bubble formation may be looked upon as a serious complicating factor." More recently End (6) says: "Rapid decompression of experimental animals causes exaggerated agglutination, and even when it is impossible to demonstrate any gas bubbles in the body, some of these animals show multiple petechial infarcts due to thrombosis or to impairment of circulation by cellular emboli may form relatively large infarcts, and this delay in forming large lesions may correspond to the delay in onset of symptoms of compressed air illness."

"Swindle has shown that an increased amount of carbon dioxide will cause an increase in the extent and duration of agglutination,

just as an increase in the amount of carbon dioxide increases the incidence and severity of compressed air illness. Alkalies and oxygen tend to prevent or diminish agglutination, just as they tend to prevent (End, 1938) or diminish the severity of compressed air illness (Behnke and Shaw, 1937). End and van Hecke (unpublished data) have been able to demonstrate a marked fall in the carbon dioxide combining power of the blood during prolonged compression, which apparently explains the fact that difficulty in decompressing increases even after complete saturation with nitrogen, due to increasing acidity of the blood, which favors agglutination. The effectiveness of alkalization in preventing and treating compressed air illness (End, 1938) may be due to the part it plays in replenishing alkali reserve of the body." * * * "While the phenomenon of agglutination described by Swindle forms a very convenient basis for a theory of compressed air illness, it must not be forgotten that bubble formation may occur as a complicating factor and if severe may dominate the clinical and post mortem picture." These observations are the first attempt in over 25 years to explain the cause of compressed air illness on any other than the mechanical nitrogen air bubble theory, and for that reason are given prominent consideration in this report.

SYMPTOMS

The usual symptoms are mentioned in a general way in most of the recent literature but there are two special subjects given more notice than ever before. One is the otological effect and the other is the bone and joint pathology of compressed air illness. Gandino (7) discusses this special condition; Crespi Reghizzi (8) reports cases of auricular disturbances in industrial caisson workers; Chiappe (9) presents a histo-pathologic study of lesions of the internal ear due to decompression; while Bertoin (10) discusses two cases of deafness due to lesions of the labyrinth as a sequel of compressed air illness. There is some confusion in the literature concerning ear involvement due to destructive processes associated with true compressed air illness as reported by the above authors, and ear conditions caused by failure to properly equalize the pressure on both sides of the drum. The latter condition is purely mechanical, is acute, usually occurs while pressure is being built up, is now termed *aerotitis media*, and is not part of the symptomatology of compressed air illness. Manigan (11) describes such cases of acute damage to the drum and middle ear under the heading of "Otological aspects of Caisson Disease"; and Requarth and Benson (12) give a general discussion of compressed air illness with special reference to the middle ear, but as their cases occurred in pressures of less than 12 pounds and the symptoms referable to the middle ear and Eustachian

tube were of an acute nature the condition must have been *aerotitis-media*.

Although the myalgias, arthralgias, and ostalgias of compressed air illness have been noted since the earliest reports, special interest now seems to be focused on bone and joint pathology, for in the 5 years covered by this report there have been nine papers on this subject exclusively whereas in the preceding 48 years there were only five such papers. Kahlstrom, Burton, and Phemister (13) review the meager literature on this phase of the condition and present four cases very completely studied and all revealing "extensive lesions * * * in diaphyses and in epiphyses of certain bones with changes in the joints, all of which appeared to be the result of massive aseptic necrosis of bone secondary to interruption of its blood supply by the liberated nitrogen gas." Rendich and Harrington (14) also report four cases showing chronic bone changes of aseptic necrosis involving the hips, shoulders, or knees, medullary calcification in the diaphyseal ends of long bones and hypertrophic arthritis. Walker (15) presents a case which he saw 25 years after having compressed air illness and says: "There can be no doubt that the lesions of the right femoral head and lower shaft, and right tibia are on a nitrogen-embolism basis." He outlines the sequence of events as follows: Nitrogen bubble—emboli—infarction—aseptic necrosis of intramedullary bone—repair by formation of fibrous wall around necrotic area—then calcification and ossification. Coley and Moore (16) say that the entire picture noted above may be explained on the basis of vascular insufficiency and nutritional disturbance and also that it is quite likely that the characteristic lesions can result from one "insult" of sufficient severity. Frank and Knoflach (17) report "match-head-sized round rarefaction in the head of the femur" in a case 1½ years after the attack of compressed air illness. Jaeger (18) reports arthritis deformans of the hip joint although in this case compensation was denied because of insufficient proof as to the causative relation between the condition and compressed air illness. Barbara and Isola (19) present the clinical roentgenological and medicolegal aspects of "chronic osteo-arthritis due to caisson disease"; Seifert (20) discusses involvement of the hip joint as a rare manifestation of "divers disease"; and Molfino (21) discusses the "arthralgic form of caisson disease."

Gotten (22) presents seven cases of neurological manifestations ranging from blindness to complete paralysis of the lower extremities and stresses the fact that it is extremely difficult to determine at times whether the condition is really due to air pressure, is hysterical or psychic, or whether the patient is a malingerer. Gerbis (23) presents a case of brain injury due to compressed air illness which he

describes in some detail. Nissen (24) presents as a rare manifestation a case of acute hemorrhagic pancreatitis; Manabe (25) presents one case of "severe caisson disease"; while Quadri (26) presents a discussion of compressed air illness with a presentation of 41 cases. Lichtenstein and Zeitlin (27) present a histo-pathological study of a case with spinal cord involvement of 25 years' duration. He had developed paralysis of both legs and had never completely regained their proper use, because of "chronic combined degeneration of the cord due to aeropathy (caisson disease) of over 25 years' duration." They say that the thoracic portion of the spinal cord is the site of prevalent involvement, and that the white matter is very much more affected than the gray. Barrat and Bastian (28) present nine cases demonstrating ocular disturbances in workmen in caissons. Gordon and Heacock (29) demonstrated by x-ray, gas in the synovial sacs of both knee joints of a worker brought out from 25 pounds pressure without adequate decompression because of a broken leg.

TREATMENT

Treatment of this condition continues to be immediate recompression and most authors agree with Fraser (30) who says, "The longer a man is kept at atmospheric pressure the more difficult will he be to treat." The breathing of oxygen or oxygen mixtures during treatment decompression is recommended by Behnke and Shaw (31) in conjunction with a special form of decompression outlined by them. More recently Yarbrough and Behnke (32) have advocated a further change in the method of treatment. They recommend that recompression be applied to one atmosphere beyond the level of relief of symptoms but that the maximum pressure be not less than 45 pounds or more than 75 pounds per square inch. At the maximum depth they recommend a stay of 30 minutes, or until symptomatic relief occurs but not for over 2 hours. The decompression treatment is started by an ascent from the maximum depth to a level of 60 feet (gage) at the rate of 25 feet per minute. "Oxygen inhalation is begun upon reaching the 60 foot level and is continued for 1½ hours, during which period the return of the patient to normal pressure proceeds * * *." This treatment was sufficient to afford permanent relief to 49 of their 50 patients.

The use of glucose is suggested by Henderson (33) who says: "10 percent glucose in normal saline intravenously, 1,000 cc. every 8 hours, appears to have great value." In motor disturbances he recommends spinal puncture as does Bienvenu (34) who treated three cases of motor disturbance by spinal puncture every third day and noted steady improvement. Rozanov (35) gives a short review of recompression in the treatment of compressed air illness.

PROPHYLAXIS

There has been but little change in the regulations designed to prevent the occurrence of compressed air illness. The medical regulations as pointed out by Singstad (36) are those usually mentioned and include the original physical examination to rule out all chronic or acute disease, the air pressure test, the periodic physical reexamination, the barring of alcoholics, maintenance of sanitary working conditions, and the attendance of a physician if the pressure is over 17 pounds. From the experimental approach to this problem Kravchinskiy and Shistovskiy (37) reported on the effect of oxygen inhalation on nitrogenous desaturation of tissues under conditions of high atmospheric pressure and a year later Behnke (38) applied the measurements of nitrogen elimination to the problem of decompression of divers. The continuous method of decompression retains some advocates although the stage method is the most generally accepted. Dorello (39) presents the "so-called mixed decompression for diving."

Oxygen continues to be recommended as the gas to be breathed in order to shorten the decompression time, for as pointed out by Behnke and Willmon (40) "It allows excess nitrogen or helium gas to diffuse from the body at a maximum head pressure." By far the most complete study of the use of oxygen to shorten the decompression time was carried out by Jones and his associates (41) in the recent construction of another New York City tunnel. A brief summary will indicate the magnitude of the work: (1) 3,884 decompressions with oxygen inhalation over a 3-month period on groups of 12 men in 3 crews. Carefully instructed and supervised. No "bends." (2) 15,904 decompressions with air in the usual way, same lock, same period of time but with 21 cases of "bends." (3) 11,904 decompressions with oxygen breathing. Large groups not instructed or supervised with 23 cases of "bends," but all were very mild and 3 were questionable. (4) At the same period as (3) there were 9,462 decompressions in ordinary air with 12 cases, but 5 were very severe. They conclude that the value of oxygen inhalation during the whole or a major portion of the period of decompression is obvious; but control and education are absolutely necessary.

As pointed out by the author (42) in a recent review, helium was suggested for use in diving as early as 1919 and much work was done with it prior to the period of this report but only in recent years has it been extensively used and reported upon. An interesting letter from Boycott (43) indicates that he had in mind its use in decompression in 1935. End (44) in 1937 pointed out the possibility of shortening the decompression time "to less than one twenty-third of the time required when compressed air is breathed." Again in 1938 (5) he pointed out the value of helium oxygen mixtures in enabling a diver to work satis-

factorily at the great depth of 420 feet. Behnke and Willmon (40) report on the successful use of a 25 percent oxygen 75 percent helium mixture in the diving operations connected with the salvage of the U. S. S. *Squalus*. Desoille (45) in a general review of the entire field recommends not only the use of oxygen and helium-oxygen to facilitate the elimination of nitrogen but also mentions the possibility of the use of hydrogen. He even suggests the elevation of the CO₂ tension in the gas phase so as to cause deep breathing and thus increase the rate of nitrogen elimination.

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A comprehensive work of 510 pages on "Deep Diving and Submarine Operations" by Davis (46) was received in this country in 1936. This work is of a general and popular nature covering primarily the mechanical aspects of diving and underwater salvage, but it also contains sections dealing with the various aspects of compressed-air illness and presents decompression tables for all depths. The book is profusely illustrated and has already been widely quoted by other British authors but as Davis is not a medical man his discussion of compressed-air illness is quite inadequate.

The remaining references which are not numbered and follow immediately after this note, are of such a general nature as not to fit logically into any of the other divisions of this review and are here presented merely for the sake of completeness.

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SULFAPYRIDINE IN GONORRHEA

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Following a rather unsatisfactory percentage of cures and too many complications with the per-oral treatment of gonococcus infection of the urethra with sulfanilamide in conjunction with local treatment, sulfapyridine therapy was instituted late in 1939. This article is a summary of our experience in a series of 22 cases.

Chemistry.—Sulfapyridine, also known as M. and B. 693 and 2 para-aminobenzene-sulfanamido pyridine, has the following formula: $\text{NH}_2\text{—C}_6\text{H}_4\text{—SO}_2\text{NH—C}_5\text{H}_4\text{N}$. Sulfanilamide is a single benzene ring with attached sulfur and nitrogen radicals. Sulfapyridine is the same sulfanilamide to which has been added a second six-sided ring, a pyridine, or nitrogen containing ring.

Pharmacology.—The pharamacology is not clearly established. It is thought that this drug acts to inhibit the growth of organisms to such an extent that the specific antibodies which the patient is able to develop can sensitize the invading organisms so that phagocytosis takes place. It is also thought that the drug exerts a bacteriostatic or bacteriocidal action on the invading organism. It is further stated that the drugs of this group act by neutralization of some metabolic or enzymatic action. After absorption it is found in the blood in both free and conjugated (acetylated) form. It is excreted only in the urine and in both free and acetylated forms, in a less rapid manner than sulfanilamide and excretion may continue for 3 to 4 days following a single dose.

It has been found by some workers (1) that the bacteriocidal action of sulfapyridine in acid urine was almost the same as in alkaline urine, differing in this respect from other sulfonamide compounds.

It has been found that treatment of pneumonia with sulfapyridine for 24 to 48 hours often alters the pneumococcus so that it becomes impossible to determine its type.

In this respect the following findings are pointed out. Since the practice of getting a daily urethral smear following institution of sulfapyridine therapy, as long as any discharge is present, we have been able to note that in many cases as the number of gonococci decreased there were structural and other changes taking place. Many organisms appeared to have expanded in size and assumed a degenerative type of appearance. The ability to stain Gram negative appeared to be lost and often on the second and third day of treatment

the few remaining organisms were almost Gram positive in their staining characteristics.

Treatment.—In our treatment a definite routine is followed with minor variations. In every case, treatment is instituted as soon as possible after the diagnosis is made.

The total dosage of sulfapyridine used is 30 to 35 grams over a period of 5 to 6 days. Treatment is routinely started at 10 o'clock in the morning at which time $1\frac{1}{2}$ to 2 grams are given with 10 grains of soda bicarbonate in capsule form. This dosage is repeated at 4-hour intervals, and the last dose given is at 10 o'clock at night. At 2 o'clock the following morning the dosage is reduced to 1 gram and so continued through the day and the next 3 days. On the sixth day a dose of one-half gram of sulfapyridine is given and treatment is discontinued at the end of the day. Soda bicarbonate grains 10 is continued with each dose of the drug during the 6 days of treatment.

In cases where the patient vomits immediately or before 30 minutes after taking the drug, the entire dose is repeated, one tablet every 5 minutes. If vomiting occurs from 30 to 60 minutes following this dosage one-half the dose is repeated; if 1 hour has passed with no vomiting there is no repetition of the drug.

During the first 24 hours of treatment 5 grains of veronal (barbital) are given at 6 p. m., 10 p. m., and 2 a. m. to combat nausea and vomiting. This is repeated the following nights as needed. Usually one or two tablets are sufficient. (See table 1.)

Before institution of treatment it is necessary to have a complete blood count, haemoglobin determination and urinalysis. Every subsequent day of treatment a white blood count, differential and urinalysis are done. As long as a discharge is present an urethral smear is made daily and stained by Gram's method. The urine is studied for red blood cells and crystal formation. About 10 days after institution of treatment, if no discharge is present, a very light prostatic massage is done to obtain material for a smear after the two-glass examination of urine has been made. This is repeated at weekly intervals for 2 or 3 weeks. Similar examinations are made at 6- and 8-week intervals. Two to three days after the treatment is discontinued a complete blood count is made and a complete urinalysis is done. In all cases where there has been a reduction of white count, the blood examination is repeated 1 month after treatment is begun. (See table 2.)

No blood determinations of sulfapyridine concentration have been made at any time.

No local or other treatment than that given in table 1 has been used in conjunction with sulfapyridine therapy with the exception of four patients who did not respond to this treatment.

Gram stains are made on urethral smears when possible throughout the course of treatment and during the 2 to 3 months follow-up treatment.

Results of treatment.—Of the 22 patients treated 13 were new cases of gonorrhea. Nine were old cases who had previously received 1 or more courses of sulfanilamide with the original infection still present.

In 17 of these patients the discharge persisted for an average of 4.2 days and organisms were present in the smears for an average of 3 days. Thereafter these patients were negative in every respect.

Three of the remaining five cases had previously been treated with sulfanilamide unsuccessfully.

Three of the five cases responded after 2 days of treatment with a cessation of discharge, but from 2 to 10 days after completion of treatment, again had a positive discharge, or were found, on routine examination, to have gonococci in the prostatic smears. A repeated course of sulfapyridine cleared one of the three again and he has remained negative to all examinations for the last 3 months.

Two did not respond to the repeated course of treatment and were then placed on daily urethral injections of 3 percent silvol. The discharge gradually cleared up and has been negative for 2½ months.

The remaining two recalcitrant cases never quite cleared of discharge or of urinary symptoms of gonorrhea during first course of sulfapyridine therapy, and so were placed on daily urethral injections of 3 percent silvol. All symptoms disappeared and urethral smears were negative for 2 months.

The urine cleared in all patients reacting favorably to the drug in 24 to 48 hours with two exceptions and no cause for the continued findings of pus and shreds in the urine in these two cases could be ascertained.

Complications.—There were no cases of epididymitis, or arthritis in this series. There was some degree of prostatic tenderness (not marked) in each of the five cases who did not respond favorably to the first course of therapy. No other prostatic involvement was noted.

The complication most noticeable was that of nausea and vomiting. It was present to a slight degree in nearly every case and was severe enough in about 25 percent of cases to make it necessary for the patient to go to bed for a period of a few hours to 1 or 2 days.

Routine urine examination revealed the presence of crystals, apparently acetylsulfapyridine, in two cases. In one case, the crystals disappeared in a few days. In the other, a moderately severe hematuria appeared on the fourth day of treatment. Treatment was discontinued, the hematuria persisted for 3 more days then disappeared.

Urinalysis revealed granular and mucous casts in this patient's urine for about 4 weeks afterward, but since the urine has been normal. This patient was negative for discharge or organisms after the third day of treatment. At the time of hematuria he complained of some pain in both kidney regions.

A mild degree of cyanosis was noted in practically every case but cleared rapidly after completion of treatment. General malaise was present during treatment in the majority of cases.

There were no changes in red cell count or haemoglobin.

In the majority of cases there was a temporary mild neutropenia and a leucopenia. The total white count dropped to slightly over 4,000 in 2 or 3 patients, but rose the following day, and continued within normal limits while yet under treatment. In most cases there was a mild decrease in neutrophils with resulting increase in lymphocytes and monocytes, but this reaction was always temporary.

Discussion.—No determination of concentration of sulfapyridine in the blood was taken on any patient in this series due to lack of facilities for such tests. Schulze (2) states that there is no relationship between the level of the free drug in the blood and the ratio of dosage to body weight. He further states no optimal blood level for free sulfapyridine is indicated. The incidence of toxic effects in his series of cases, he feels, cannot be shown to be directly related to the free blood sulfapyridine level.

Other men (3) have found no need of delaying the beginning of treatment after discharge is apparent. This corresponds with our findings. These men found no blood changes in any case of cyanosis nor depression of erythroblastic activity at any time. They also state that some cases showed a slight depression of total white count but that a normal white count soon followed the cessation of treatment. It has been our experience that following a diminution of white count there followed within 24 to 48 hours an increase to slightly above the average normal for total white count, and a return to a normal differential while still under treatment.

Carroll, Shea, and Pike (4) describe a case of pneumonia treated with sulfapyridine in which a complete anuria resulted from filling of the kidney pelves with acetylsulfapyridine crystals. This was relieved by inserting catheters cystoscopically and lavaging the pelves with warm normal saline or sterile water. These crystals seen in large groups are not opaque to x-ray and flat Roentgenograms are of no value. They state that hematuria noted in this form of treatment is probably due to minute traumatism of the mucous membranes by crystals and is of no grave consequence. They further believe that stopping treatment and forcing fluids is all that is indicated in handling this complication. Our case with hematuria seems to bear out these assumptions.

It is our belief that with the findings concerning the relation of blood levels to the efficacy of the drug it is likely that better results will be obtained in this treatment if fluids are forced rather than limited. Due to the nausea and vomiting and possible dehydration that may complicate some cases it is best to force fluids. Gross, Cooper, and Lewis (5) believe that increased fluids will prevent to some extent precipitation of the crystals in the urine. They state that the limited solubility of sulfapyridine (1:1,000 at room temperature) and even greater insolubility of its acetylated derivative, makes precipitation in the urine very likely, particularly if the fluid intake of the patient is low or if there is a marked water loss. Their analysis of urinary calculi in experimental animals given sulfapyridine have shown most calculi to vary in size from that of sand particles to 3 x 3 x 4 mm. Crystals were spiculated and consisted of radiating crystallizing needles and plates. Analysis of the calculi showed the presence of 6.4 percent sulfapyridine and 64.1 percent acetylsulfapyridine.

Long and Wood (6) favor the alkalization of urine to prevent acetylsulfapyridine precipitation. It may be that the routine giving of fairly large doses of soda bicarbonate in our own series explains the presence of crystals in the urine of only two patients. However, the soda was given in this series as was the barbitol, primarily to eliminate nausea.

It was reported (7) in a recent series of cases, treated by various measures, that sulfapyridine induced a more immediate relief of symptoms than did sulfanilamide but that late relapses (often 6 months) were more numerous after the former drug. The cases in our series do not date back more than 6 months but most of them are reaching an approximation of that time and there has been no indication from carefully followed-up routine examinations that any recurrences have occurred.

SUMMARY

1. Sulfapyridine clears the urine and stops discharge quickly in majority of cases of gonorrhea treated regardless of stage of disease.
2. No local treatment is needed in conjunction with those cases that show a marked response to this type of treatment.
3. Complications noted in order of frequency are; Nausea and vomiting, anorexia, cyanosis, leucopenia, neutropenia, and hematuria.
4. The more physical labor done by the patient while under treatment, the more marked the nausea, the vomiting, and malaise.
5. Extension of the infection with resulting prostatitis, epididymitis, arthritis, or other such complications has been practically nil.
6. It is believed that while slightly smaller beginning doses, than those given in this series will effect the same decrease in symptoms with

less nausea, nevertheless, the rather large dose of the drug used throughout this series is possibly responsible for the favorable results obtained.

7. Blood concentration level determinations of the drug are not of great importance.

8. This drug should be given only to patients who can be well controlled as careful blood counts and urine examinations should be made daily or every other day throughout the treatment.

9. Forcing of fluids is probably of value during this treatment.

10. All factors considered, sulfapyridine has been the most satisfactory drug we have yet used in treatment of acute and chronic gonococcus infection of the urethra.

TABLE 1.—*Dosage chart*

[A=Sulfapyridine. B=Sodium bicarbonate. C=Barbital]

Day of treatment	2 a. m.	6 a. m.	10 a. m.	2 p. m.	6 p. m.	10 p. m.
1.....	CBC, Hgb., urinalysis, gram stain of urethral smear.		A. Grams. 2 B. Grains. 10	A. Grams. 2 B. Grains. 10	A. Grams. 2 B. Grains. 10 C. Grains. 5	A. Grams. 2 B. Grains. 10 C. Grains. 5
2.....	A. Grams. 1 B. Grains. 10 C. Grains. 5	A. Grams. 1 B. Grains. 10	A. Grams. 1 B. Grains. 10	A. Grams. 1 B. Grains. 10	A. Grams. 1 B. Grains. 10 C. Grains. 5	A. Grams. 1 B. Grains. 10 C. Grains. 5
3.....	A. Grams. 1 B. Grains. 10	A. Grams. 1 B. Grains. 10	A. Grams. 1 B. Grains. 10	A. Grams. 1 B. Grains. 10	A. Grams. 1 B. Grains. 10 C. Grains. 5	A. Grams. 1 B. Grains. 10 C. Grains. 5
4.....	A. Grams. 1 B. Grains. 10	A. Grams. 1 B. Grains. 10	A. Grams. 1 B. Grains. 10	A. Grams. 1 B. Grains. 10	A. Grams. 1 B. Grains. 10 C. Grains. 5	A. Grams. 1 B. Grains. 10 C. Grains. 5
5.....	A. Grams. 1 B. Grains. 10	A. Grams. 1 B. Grains. 10	A. Grams. 1 B. Grains. 10	A. Grams. 1 B. Grains. 10	A. Grams. 1 B. Grains. 10 C. Grains. 5	A. Grams. 1 B. Grains. 10 C. Grains. 5
6.....	A. Grams. 1/2 B. Grains. 10	A. Grams. 1/2 B. Grains. 10	A. Grams. 1/2 B. Grains. 10	A. Grams. 1/2 B. Grains. 10	A. Grams. 1/2 B. Grains. 10	A. Grams. 1/2 B. Grains. 10

TABLE 2.—*Showing relation of blood and urine examinations to treatment*

Sixth day of treatment.....	Completion of dosage.
Ninth or tenth day of treatment.	CBC, Hgb., urinalysis, 2 glass test, examination of prostatic and urethral smears.
Sixteenth day of treatment...	2-glass test, examination of urethral or prostatic smear, blood examination in special cases.
Twenty-third day of treatment.	Do.
Thirtieth day of treatment....	Do.
Forty-fifth day of treatment..	Do.
Sixtieth day of treatment.....	Do.

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SULFATHIAZOLE TREATMENT OF GONOCOCCAL INFECTION OF AMBULATORY CASES ABOARD SHIP

SUPPLEMENTARY REPORT

By Lieutenant (Junior Grade) E. E. Metcalfe, Medical Corps, United States Navy

Introduction.—Having completed a series of cases of gonococcal infection of the urethra by means of sulfapyridine it was decided to conduct a comparative study with sulfathiazole.

At the onset of this study very little material had appeared in the literature on sulfathiazole in the treatment of gonorrhea making it necessary to carry out considerably more laboratory work than is now indicated in treating these cases.

Analysis of cases treated.—Seventy cases of gonorrhea were treated; 6 cases had been previously treated with sulfanilamide; 5 cases had been treated with sulfapyridine, and 8 cases had received both sulfanilamide and sulfapyridine without satisfactory results; 51 cases had received no previous form of therapy. Clinically the cases may be grouped as follows:

Anterior urethritis.....	45
Posterior urethritis.....	11
Anterior and posterior urethritis.....	6
Posterior urethritis and prostatitis.....	8

Time elapsed since onset of infection:

1 to 10 days.....	42
11 to 20 days.....	10
21 to 90 days.....	18

The cases averaged 21.8 years and were in excellent physical condition; four were also under active antiluetic treatment.

Transcripts were made from the records of those having had sulfanilamide or sulfapyridine to act as a basis for comparative study with sulfathiazole.

Method.—All cases were given a complete physical examination to determine the nature of the existing condition and to rule out any pathology that might contraindicate this method of treatment.

Laboratory work consisting of haemoglobin, red cell count, white count, differential, sedimentation time, and routine urinalysis was

done at the beginning of treatment and again at the termination. Haemoglobin and white count and urinalysis for albumin and blood were made on alternate days during the treatment. All men were examined daily and questioned for complications. Daily two-glass urine tests were done.

Treatment.—The regime of the first 30 cases consisted of 4 grams of sulfathiazole given with 4 grams of sodium bicarbonate in 4 equally divided doses the first day; 2 grams of the drug with 2 grams of sodium bicarbonate in 4 equally divided doses were given for the 9 succeeding days. In the last 40 cases the sodium bicarbonate was omitted with no untoward results. No form of local treatment was used. The use of liberal quantities of fluids was encouraged. Medication was better tolerated $\frac{1}{2}$ to 1 hour after meals. In 2 cases therapy was continued over the 10-day period. In the first 35 cases activity was restricted; the remaining cases assumed their routine duties.

Discussion of toxicity and reactions.—As sulfathiazole is more rapidly metabolized and undergoes less conjugation than sulfapyridine, toxic action is more readily controlled. According to Long, Haviland, and Edwards (2), 80 to 90 percent may be recovered in the urine in 24 hours. This more rapid absorption and excretion undoubtedly accounts for its superiority over sulfanilamide and sulfapyridine in numerous ways.

Reinhold, Flippin, and Schwartz (3) report vomiting occurring in about 10 percent of their cases. With dosage used in our series no vomiting was experienced, but 25.7 percent experienced nausea the first 2 days of administration of the drug. No special measures were necessary to control nausea and the drug was not discontinued. Cyanosis was not observed in any of our cases.

Haviland and Long (4) observed 10 cases with skin eruptions in a group of 78 cases. They divide the rashes into 3 main groups: Maculopapular, urticarial, and a rash resembling erythema nodosum. We observed 2 cases of skin eruption in our series, one in a fireman who was on duty in the boiler room and another in a cook in the galley. These rashes were maculopapular in type. Both men were relieved from their duties and rashes cleared in 24 hours. Medication was not discontinued.

No attempt was made to prevent exposure to sun during the administration of the drug as was done with sulfanilamide or sulfapyridine; in fact during most of the study, the ship was cruising in tropical waters.

Mitchell, Greig, and Uren (5) noted headache in 2 cases in a series of 12. We had 6 cases that experienced a feeling of fullness in the frontal regions which was relieved by fresh air.

Pepper and Horack (6) have reported that sulfathiazole is re-crystallized in the kidney tubules whereas sulfapyridine crystallized most frequently in the renal, pelvis, and ureters. Reinhold, Flippin, and Schwartz (3) report that kidney function was temporarily impaired in a varying degree in nearly all of their patients. Knoll and Cooper (7) report a case of hematuria and albuminuria with sulfathiazole administration.

These observations demonstrate the need for routine urinalysis and renal function tests at the completion of the course of therapy. Our microscopic and benzidine tests done on all cases on alternate days revealed no evidence of hematuria.

Burkholder and Bang (8) report cases of elevation of temperature in their series. We had no elevation in temperature above 99.8° in our work and did not stop drug in these cases.

Results.—Upon the completion of treatment all men who were apparently cured were given prostatic examinations, and the smears of prostatic secretion were stained for examination. Results of the two-glass urine tests were recorded. On the following day sounds were passed as far as the bulbomembraneous junction and the canal massaged over it.

Of the apparently cured, all of whom received sounds the day following their prostatic massage, none developed a urethral discharge afterward or any subsequent symptom of prostatitis as evidenced by the two-glass test.

SUMMARY OF RESULTS

1. Twenty five and seven-tenths percent experienced nausea (75.7 percent experienced nausea with sulfapyridine).
2. Average drop in white count during course of treatment, 1,227 (average with sulfapyridine 2,514).
3. The drug had no effect on haemoglobin.
4. Average drop in sedimentation time, 2.62 mm.
5. Eleven and six-tenths percent experienced headaches.
6. Two cases developed a maculopapular rash that disappeared in 24 hours.
7. No complications developed during treatment.
8. Average dosage of drug received, 338 grains.
9. Average number of days treated, 10.1.
10. Average days for cessation of discharge, 3.
11. Ninety-seven and fourteen one-hundredths percent apparently cured.
12. Average number of days for clearing of two-glass test, 5.
13. Infected 1 to 10 days, 95.2 percent cured; infected 11 to 20 days, 100 percent cured; infected 21 to 90 days, 100 percent cured.

DISCUSSION

The dosage used and the resultant blood level of sulfathiazole reached was not known due to inadequate facilities aboard ship for carrying out these determinations. However, Mahoney, Wolcott, and Van Slyke (9) employing a somewhat similar dosage schedule to the one used by us, found marked individual variations in the blood levels.

In comparing the reactions of sulfathiazole to those met in the use of sulfapyridine and sulfanilamide, it is apparent that sulfathiazole is less toxic than either.

The rapid cessation of the discharge and the clearing of the two-glass test in early cases demonstrated the efficacy of the drug over sulfapyridine where it was apparent that better results were obtained if a time interval were allowed for active immunity to be established.

The previous treatment using sulfanilamide or sulfapyridine evidently had no effect on the results obtained using sulfathiazole as those who had previously failed to respond to treatment with these drugs responded to sulfathiazole.

The two failures experienced in the series did not respond to a second course of sulfathiazole and were placed on local therapy.

A very interesting experience with this drug may be cited to suggest its use as a prophylactic. Fifteen men who were exposed to known prostitutes without condoms and who did not use sanitubes following these exposures were given 4 grams of sulfathiazole the next day in 4 equally divided doses. The following 2 days they received 2 grams in 4 equally divided doses. They did not develop any urethral discharge. It is assumed that for prophylaxis the drug should be given 24 hours before exposure and for the 2 days following.

CONCLUSIONS

1. Seventy cases of gonorrhea were treated with sulfathiazole with 97.14 percent apparent cures.

2. Sulfathiazole may be given in ambulatory cases of gonorrhea with a more reasonable margin of safety than sulfanilamide or sulfapyridine.

3. Sulfathiazole is superior to sulfapyridine in the treatment of gonorrhea.

4. Ambulatory doses as given apparently can maintain the blood sulfathiazole at a level sufficiently high to effect a cure.

5. A suggestion for its use prophylactically has been mentioned.

6. Toxic symptoms experienced in our cases were negligible.

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EVALUATION OF THE USE OF SULFONAMIDES IN THE TREATMENT OF GONORRHEA ¹

By Lieutenant Commander French E. Moore, Medical Corps, United States Navy

The treatment of gonorrhea by the use of sulfanilamide and its derivatives, sulfapyridine and sulfathiazole, is so gratifying that it almost becomes pleasure to treat these cases. It is indeed a far cry from the days before 1937 when our venereal wards were filled with old recurring gonorrheal cases.

Results first reported following the use of sulfanilamide were nearly as favorable as those now seen in the literature of sulfapyridine and sulfathiazole. As we continued to use sulfanilamide, however, the rate of cure dropped, until now, instead of curing 80 to 85 percent of our cases, the rate is 55 to 60 percent, when the use of the drug is combined with local treatment. However, this will not prove to be the case with sulfapyridine and sulfathiazole as our present experience indicates that the rate of cures with these drugs will be much higher, with a decrease in the number of sick days per case.

Parsons (1) has recently shown the remarkable improvement in the treatment of all types of gonorrheal infections since the use of sulfanilamide. Clinical trial has already shown both sulfapyridine

¹ Presented before the annual meeting of the Territory of Hawaii Medical Society, May 2, 1941.

and sulfathiazole to be superior to sulfanilamide, so that we can expect an even lower rate in complications, less days on the sick list per case, and probably a lower incidence in 1941 and 1942.

From the recent literature comes several reports on sulfathiazole. First, Culp (2) reports 21 cases of acute gonorrhea in the male who completed a full course of treatment with 80.9 percent of cures. Seventeen of these cases showed no recurrences. Four cases showed negative smears after 24 hours' treatment and 7 cases after 48 hours. Of the 4 patients that failed to respond to sulfathiazole treatment, 2 had received the drug 10 and 12 days, respectively, a third case also failed to respond to sulfanilamide or sulfapyridine, and in the fourth case the drug was discontinued because the hemoglobin was reduced to 77 percent.

Burkholder and Bangs (3) reported 100 cases also treated by sulfathiazole with 92 percent cured. All of these patients were hospitalized throughout the treatment. They started treatment with an initial dose of 4 to 5 gm., and then continued with 1 gm. every 4 hours for 14 days. The blood concentration ranged from 2 to 18 mg. per 100 cc., with an average of 6 mg. per 100 cc. They reported 13 reactions among these 100 patients, 6 of these had urticaria with painful joints, 5 mixed bulbar conjunctivitis, 1 had nausea, 5 had febrile actions, and 1 had a transitory leukopenia of 2,700 white blood cells.

Knight, Latkowski, and Ukle (4) report 50 cases which were followed, and 96 percent of which were reported as cures. The average duration of treatment was 8 days with the drug dosage averaging 28 gm. Two cases were reported as failures. They reported the majority of the cases as receiving 3 gm. daily for 8 days. Toxis reactions occurred in 11.5 percent of the patients, and consisted of, headache in 3 cases, nausea in 1 case, and vertigo in 1 case.

Mahoney (5) (et al.) report 47 cases of unselected male patients with acute gonorrhea with 91.5 percent of cures. Their patients received a dosage of 4 grams the first day and 2 grams daily for 5 to 11 days thereafter. The maximum amount given was 42 gm. over a period of 10 days and the minimum was 11 gm. in 5 days. They also report that evidence of toxicity was negligible.

Ballenger (6) (et al.) reported 45 cases in which injections of silver protein were given in addition to sulfathiazole in male patients having acute gonorrhea. Immediate cure followed in 5 patients who presented themselves within 2 days after onset of symptoms. In the other cases discharge disappeared within the average time of 12 days. They reported that a few patients had nausea and vomiting while taking sulfathiazole but 20 percent of their cases on sulfapyridine had gastric upsets.

Mitchell, Greig, and Uren (7) treated 12 cases of acute and chronic gonorrhea with sulfathiazole. Eleven of the patients responded to sulfathiazole therapy and all of these were free of gonorrheal discharge within 3 days. The rate of cure in this small series was 91.7 percent as compared to 87 percent in a series of 252 cases treated with sulfapyridine.

The results of the treatment of gonorrheal patients at the United States Naval Hospital, Pearl Harbor, T. H. were reviewed since August 1939. Sulfanilamide was used in 87 cases from August 1939 to February 1940. These were routine consecutive admissions, the types of infections being as noted in chart. In this group there were 7 recurrences and 31 failures. Percentage cured was 59.8. Average time on the sick list was 37 days. Dosage of sulfanilamide was 80 grains daily for 2 or 3 days, 60 grains daily for 7 to 10 days, and 40 grains for 8 to 12 days. Local treatment was also used in all sulfanilamide cases plus prostatic massages when indicated. The length of time until discharge ceased was 9.6 days.

Sulfapyridine was used from February 1940 to August 1940 on 59 consecutive patients admitted with various types of gonorrheal infections. As noted in the chart, 31 cases treated with sulfanilamide failed but these were successfully treated with sulfapyridine. The percentage of cures with sulfapyridine was 88.3 percent with 2 failures and 5 recurrences. Treatment consisted of 60 grains of sulfapyridine daily with an average total dosage of 930 grains. No local treatment was given in this group. The average number of sick days was 28 and the average length of time until discharge ceased was 4.7 days.

Starting in August 1940 sulfathiazole therapy was used. There have been 80 cases treated to the present time, March 1, 1941. The average time on the sick list was 15.5 days. There have been 5 recurrences all of which have responded to a second course of therapy.

Two failures in this group also failed to respond to sulfanilamide and sulfapyridine. The percentage cured with sulfathiazole was 91.3 percent. The daily dosage was 60 grains with an average of 805 grains per case. The average length of time until discharge ceased was 2.3 days.

A follow-up questionnaire was sent to all patients treated with sulfanilamide and sulfapyridine and in spite of the many transfers 75 percent of replies were received. The number of recurrences following use of these drugs might have been a little higher than noted in the chart. However, we have personally checked 92 percent of our cases treated with sulfathiazole for an average of 6 weeks following their discharge from treatment and found no recurrences except as noted in accompanying chart.

Blood studies.—In 26 cases receiving sulfapyridine, dosage 60 grains daily, the blood concentration ranged from 1 to 5 mgm. per 100 cc. In the group treated with sulfathiazole, also on a dosage of 60 grains daily, the blood levels ranged from 1 to 4.0 mgm. per 100 cc. of whole blood. No relation was noted between the blood concentration and the response made by the individual patient. As no effort was made to restrain fluids, our blood concentrations were never very high. The patients receiving sulfathiazole or sulfapyridine were instructed to increase their fluid intake.

Blood counts and hemoglobin estimations were done frequently on all cases included in this report. In the sulfanilamide group, 19 percent of the patients showed a decrease in hemoglobin and in both white and red cell counts. None of these changes, however, were considered severe enough to cause discontinuance of the drug. The lowest hemoglobin estimation was 68 percent Talquist; lowest white blood count 4,300 and lowest red count 3,500,000.

With sulfapyridine, the lowest hemoglobin was 80 percent Talquist; lowest white count was 3,500 and the lowest red count was 3,800,000. The white blood cells returned to normal 3 days after withdrawal of the drug.

The blood studies of sulfathiazole cases revealed no abnormalities except in one patient who received the drug 28 days. In this case, frequent blood studies revealed a gradual decrease in hemoglobin and red count but none in white count.

Reactions.—Only 1 reaction was noted among 80 patients receiving sulfathiazole. This was a generalized erythema which appeared on the eighth day of treatment and subsided within 24 hours following withdrawal of the drug.

Of the sulfapyridine group, there were 15 skin reactions in 59 cases; 6 were a generalized erythema and 9 were urticarial in type. These all subsided upon withdrawing the drug. In addition, 12 percent of the patients receiving sulfapyridine complained of slight nausea and headache.

There were 17 reactions in 87 patients on sulfanilamide; 7 of these were urticarial and 10 were erythemas. In the sulfanilamide group 14 percent had moderate gastric upset and headache but not sufficient to cause discontinuance of the drug.

Before discharging any of the above patients to duty as cured, three negative urethral smears and one negative prostatic smear were obtained. In all doubtful cases one urethral and one prostatic negative culture was required prior to discharge.

SUMMARY

1. Our experience with 80 cases of gonorrheal infections has shown that sulfathiazole is a much more satisfactory drug to use than either

sulfanilamide or sulfapyridine. The percentage of cures has been higher while the average time on sick list was nearly 45 percent less than with sulfapyridine and 60 percent less than with sulfanilamide.

2. There was only one reaction following the use of sulfathiazole as compared with 15 in the sulfapyridine and 17 in the sulfanilamide groups.

3. Blood counts on patients receiving sulfathiazole revealed no changes, while with sulfapyridine and sulfanilamide frequent blood studies are necessary.

4. No relation was found between the blood concentration and the response made by the patient with either sulfapyridine or sulfathiazole.

TABLE 1.—*Results of treatment using sulfanilamide, sulfapyridine, and sulfathiazole*

SULFANILAMIDE—USED EXCLUSIVELY FROM AUGUST 1939 TO FEBRUARY 1940

Type of infection	Number of cases	Average number of sick days	Average dosage in grains	Number of recurrences	Number of failures	Skin reactions		Percentage cured
						Urticaria	Erythema	
Anterior.....	34	28	1.025	5	8	3	3	61.8
Ant.-post.....	32	33	1.215	2	13	1	4	59.4
Epididymitis.....	10	41	1.590	-----	5	1	2	50.0
Ac. prostatitis.....	7	40	1.725	-----	4	2	1	43.8
Arthritis.....	4	43	2.000	-----	1	-----	-----	75.0
Total.....	87	1 37	1 1.510	7	31	7	10	1 59.8

SULFAPYRIDINE—USED EXCLUSIVELY FROM FEBRUARY 1940 TO AUGUST 1940

Anterior.....	24	22	585	3	-----	3	1	87.5
Ant.-post.....	16	24	610	2	2	2	2	75.0
Epididymitis.....	7	28	780	-----	-----	3	2	-----
Ac. prostatitis.....	9	31	910	-----	-----	1	1	-----
Arthritis.....	3	35	1,250	-----	-----	-----	-----	-----
Total.....	59	1 28	1 930	5	-----	9	6	1 88.2

SULFATHIAZOLE—USED EXCLUSIVELY FROM AUGUST 1940 TO MARCH 1941

Anterior.....	30	13	585	3	0	1	-----	90.0
Ant.-post.....	28	15	770	2	0	-----	-----	92.8
Epididymitis.....	10	16	840	0	2	-----	-----	-----
Ac. prostatitis.....	12	18	910	0	0	-----	-----	-----
Total.....	80	1 15.5	1 805	5	2	1	-----	1 91.3

1 Average.

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COLLAPSE THERAPY OF PULMONARY TUBERCULOSIS¹

RESULTS WITH PNEUMOPERITONEUM

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In a preliminary report (1) the indications for pneumoperitoneum in the collapse therapy of pulmonary tuberculosis were discussed. The anatomical elevation of the diaphragmatic leaves and the resulting pulmonary compression were also described. After 9 months of clinical trial definite x-ray evidence can now be presented showing the closure of pulmonary cavitation and the improvement of exudative lesions in the pulmonary parenchyma. Again it must be emphasized that artificial pneumothorax collapse of the lung is still the operation of choice. The most complete and satisfactory type of pulmonary compression is obtained by this method. It is only if this method fails, or when some other contraindication prevents its use, that pneumoperitoneum has been used. Observation has demonstrated that the pulmonary compression resulting from pneumoperitoneum is greatly enhanced by a preliminary phrenic paralysis. In the series of cases studied pneumoperitoneum was found to be only about 25 percent effective without a preliminary phrenic crush. A unilateral phrenic paralysis in combination with pneumoperitoneum will in fact demonstrate an increase as high as 8 cm. in the elevation of the paralyzed diaphragmatic leaf as compared with the opposite unparalyzed side. The combined procedures of pneumoperitoneum and phrenic paralysis are so effective because they accomplish two definite objectives, both of which have been demonstrated to play an important role in pulmonary collapse therapy. The elevated paralyzed diaphragmatic leaf can compress the lung to approximately one-third its total volume as well as eliminating the diaphragmatic respiratory excursions, another vital point in immobilizing the lung. It is these two factors, compression and immobilization as provided in a combined phrenic paralysis with pneumoperitoneum, that result in the elimination of the tuberculous process. The direct result of this compression is identical with that accomplished by artificial pneumothorax, namely the drainage of inflammatory exudates from both the pulmonary cavities and the entire bronchial tract. The phrenic paralysis results in immobilization and

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functional rest of the lung. The end result of this compression and immobilization is eventually absorption and fibrosis.

It is our purpose to present a series of selected cases of pulmonary tuberculosis, treated with pneumoperitoneum, in which clinical progress, supported by Roentgenological evidence of healing, has definitely occurred. It has already been pointed out that pneumoperitoneum as a means of inducing pulmonary collapse is in itself not entirely efficient. In the cases reported below, pneumoperitoneum has been used in conjunction with phrenic paralysis and artificial pneumothorax in both unilateral and bilateral pulmonary disease. These various combinations of collapse therapy have been so indicated.

RESULTS

I. EXTENSIVE BILATERAL DISEASE

A. Pneumoperitoneum combined with bilateral phrenic paralysis

F. C. C. admitted August 29, 1939. Physical examination of chest revealed: Expansion equal but limited bilaterally. Tactile fremitus increased both right and left upper lobes. Numerous subcrepitant rales present on inspiration following expiratory cough. Pectoriloquy is present over this same area.

Sedimentation index 22 mm. Sputum positive for *M. tuberculosis*.

Chest x-ray August 31, 1939: There is a caseous pneumonic exudate infiltrating right apex and upper five right anterior interspaces. Cross infection has occurred resulting in a pneumonic exudate infiltrating left apex and upper four anterior interspaces. Cavities are noted first and third right anterior interspaces. Impression: Pulmonary tuberculosis bilateral, chronic, far advanced, with multiple cavitation.

Pneumoperitoneum was instituted September 8, 1939. A right phrenic crush was done September 22, and a left phrenic crush was done October 5. Pneumoperitoneum has been continued with weekly refills of 1,000 cc. of air.

Chest x-ray January 9, 1940: The cavity in the right upper lobe is somewhat smaller and there is clearing of the infiltration in this region. Clearing is also noted in the left upper chest. Each diaphragm is elevated considerably by pneumoperitoneum. For additional clinical data see chart 1.

B. Pneumoperitoneum combined with unilateral phrenic paralysis

W. J. M. admitted February 7, 1938. Physical examination of the chest revealed: Expansion markedly limited bilaterally. Tactile fremitus increased right and left upper lobes. Impaired percussion resonance right and left upper lobes. Numerous subcrepitant rales are elicited over entire lung fields on inspiration following expiratory cough. Bronchophony is pronounced right and left upper lobes.

Sedimentation index 18 mm. Sputum positive for *M. tuberculosis*.

Chest x-ray February 1, 1939 (fig. 1): There is an infiltration of caseous pneumonic exudate throughout the entire right upper lobe to the level of the third rib, viewed anteriorly. There is a downward extension to the right middle and lower lobes. There is a cross infection to the entire left upper lobe from the apex to the level of the fourth interspace viewed anteriorly, with downward extension to the left lower lobe. Impression: Tuberculosis, pulmonary, chronic, far advanced.

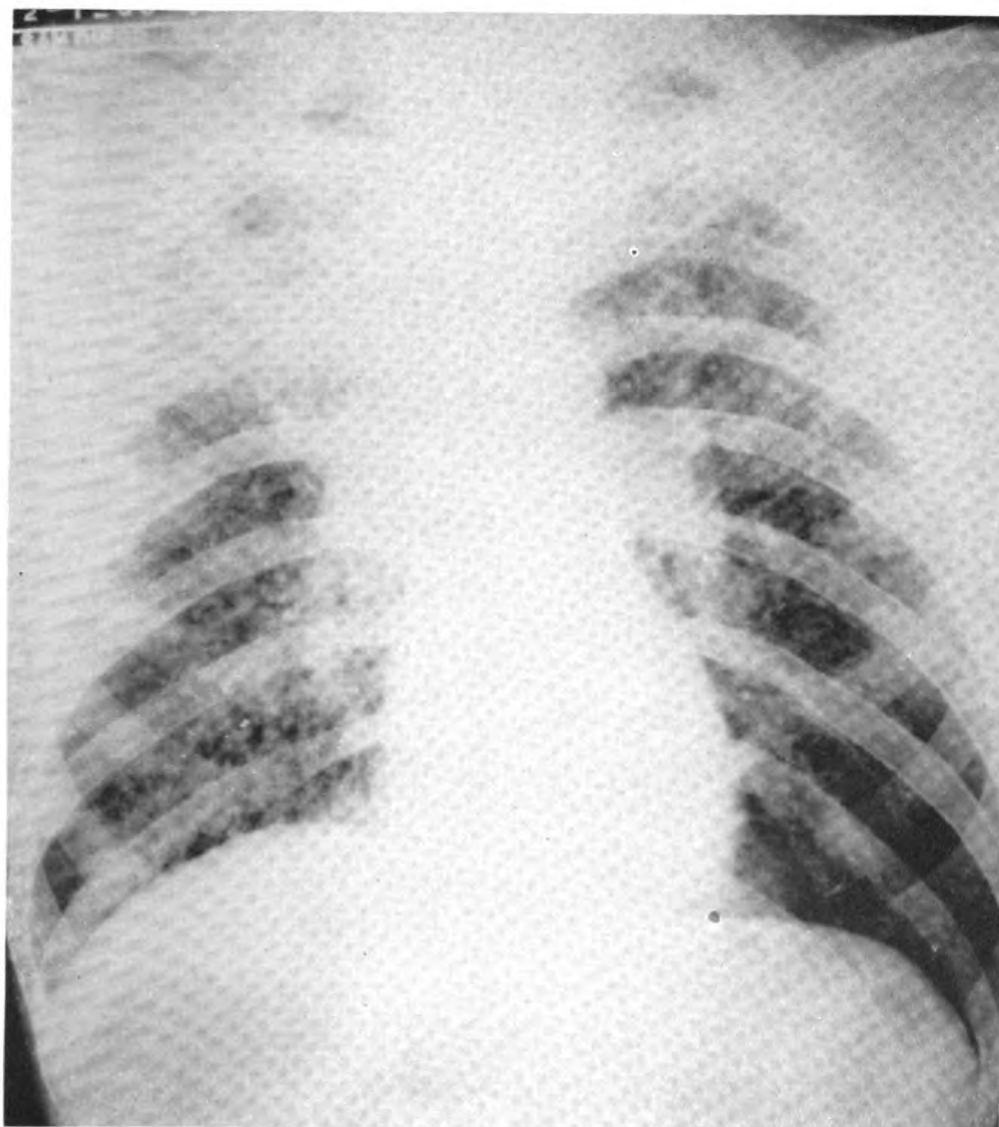


FIGURE 1.—W. J. M.—BEFORE OPERATION.

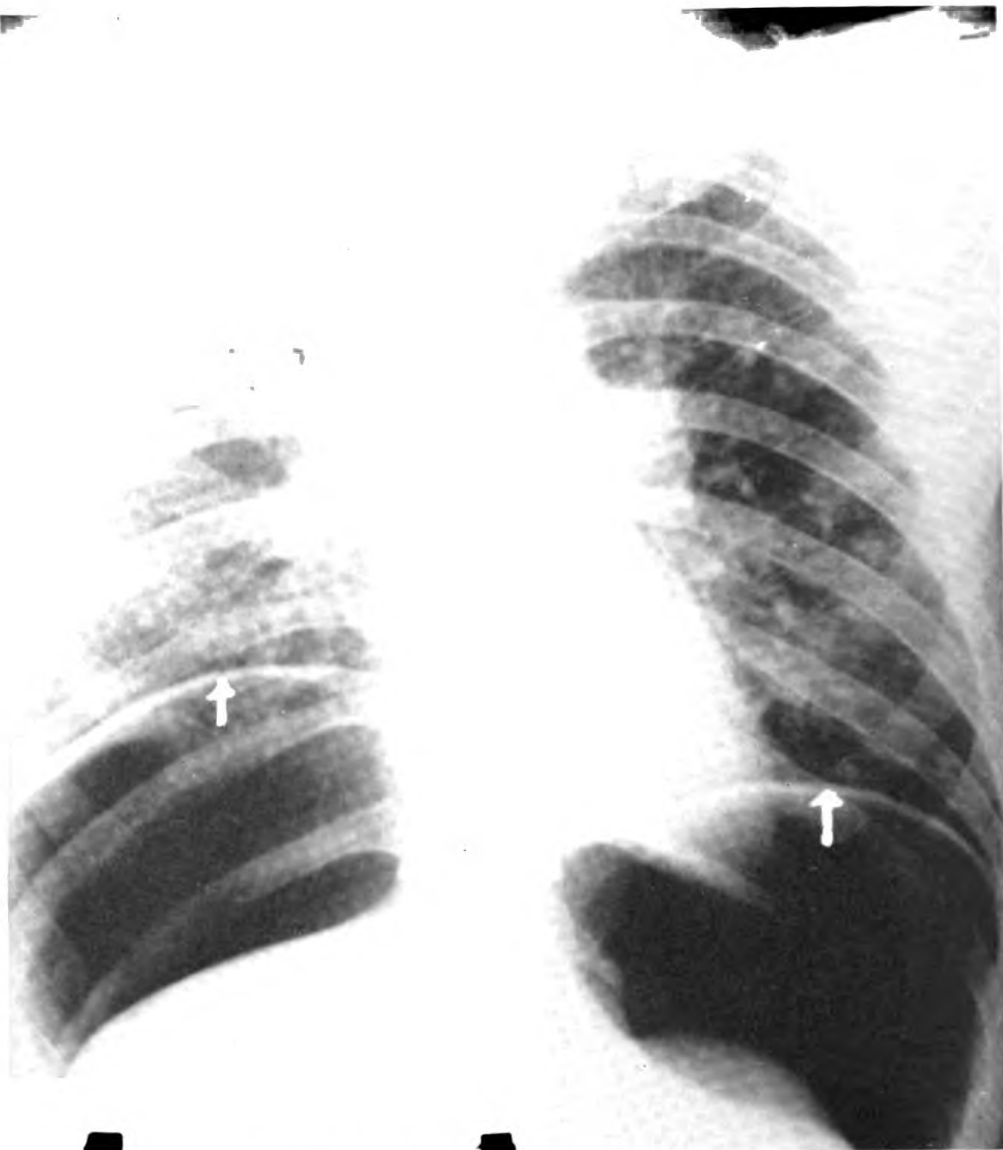


FIGURE 2.—W. J. M.—AFTER OPERATION.

A right phrenic crush was done June 8, 1939. Pneumoperitoneum was instituted June 6, and has been continued with weekly refills of 1,000 cc. of air.

Chest x-ray September 14, 1939 (fig. 2): The extensive caseous pneumonic infiltration in the left upper lobe has undergone almost complete absorption. The process in left lower and middle lobes has been replaced considerably by fibrosis. The infiltration in the right upper lobe has likewise undergone considerable fibrosis. Resolution of the process in the right base has also occurred. Each diaphragm is elevated considerably by pneumoperitoneum. For additional clinical data see chart 1.

C. Pneumoperitoneum combined with unilateral pneumothorax and contra lateral phrenic paralysis

W. F. B. admitted March 21, 1939. Physical examination of the chest revealed: Expansion is limited right upper lobe. Tactile fremitus is increased right upper chest. Impaired percussion resonance is noted right upper and middle lobes. Numerous subcrepitant rales are elicited on inspiration following expiratory cough over the right upper and middle lobes. Amphoric breathing is present over the mid portion of the left lower lobe posteriorly.

Sedimentation index 22 mm. Sputum positive for *M. tuberculosis*.

Chest x-ray March 23, 1939 (fig. 3): The right upper and middle lobes show a rather extensive bronchopneumonic infiltration. The apices and intralobar pleura are thickened. The left lung shows a cavity approximately 4.2 cm. in diameter at the third interspace, midclavicular line. There is a fluid level in this cavity. There is some exudation surrounding this cavity. Impression: Tuberculosis, pulmonary, chronic, with cavitation, probably advanced.

Pneumothorax collapse of right lung was initiated March 27, 1939, and has been continued with weekly refills of 500 cc. of air. A left phrenic crush was done April 11. Pneumoperitoneum was instituted July 4 and has been continued with weekly refills of 1,000 cc. of air. For additional clinical data see chart 1.

The chest x-ray of January 9, 1940 (fig. 4): There is approximately a 25 percent collapse of the entire right lung. The pneumonic process in the left lower has undergone considerable absorption. The cavity in the left lower lobe has almost completely closed. Each diaphragm is considerably elevated by pneumoperitoneum. The elevation is more marked on the left side.

E. B. Y. admitted April 7, 1939. Physical examination of chest revealed: Expansion equal but limited bilaterally. Tactile fremitus increased both right and left upper lobes. Impaired percussion resonance is noted at both upper lobes and left base. Subcrepitant rales are elicited on inspiration following expiratory cough throughout entire lungs fields.

Sputum positive for *M. tuberculosis*. Sedimentation index is 18 mm.

Chest x-ray April 10, 1939: There is a caseous bronchopneumonic exudation infiltrating both lungs from apex to base. This process has undergone some fibrosis at left top. Extensive cavitation is not apparent. Impression: Tuberculosis, pulmonary, chronic, active, far advanced.

A right pneumothorax collapse was induced April 20, 1939, and has been continued with weekly refills of 500 cc. of air. A left phrenic crush was done May 15. Pneumoperitoneum was initiated June 6 and has been continued with weekly refills of 500 cc. of air.

Chest x-ray November 21, 1939: The bronchopneumonic exudation infiltrating both lungs has undergone considerable resolution and fibrosis. Partial collapse of the right lung is present. Marked bilateral pneumoperitoneum is seen. For additional clinical data see chart 1.

II. UNILATERAL DISEASE

A. *Pneumoperitoneum combined with unilateral phrenic paralysis*

C. P. admitted January 20, 1939. Physical examination on admission revealed: Expansion is markedly limited right upper lobe. Tactile fremitus is increased somewhat in the right upper lobe. Impaired percussion resonance is noted over the right apex. Cavernous breathing is present over the right upper lobe anteriorly. Pertoriloquy is elicited on whispered voice over this same area.

Sedimentation index 17 mm. Sputum positive for *M. tuberculosis*.

Chest x-ray on January 17, 1939 (fig. 5): There is a large cavity measuring 2.5 x 5.6 cm. in the apex of the right upper lobe, extending from the second to the fourth interspace viewed anteriorly. Except for a small calcified area in the left lung at the level of the fifth interspace remainder of the lung fields are essentially negative.

A right phrenic crush was done January 26. Pneumoperitoneum was induced March 8 and has been continued with weekly refills of 1,200 cc. of air.

Chest film on November 28, 1939 (fig. 6): There is a considerable amount of air in the peritoneal cavity and a marked elevation of the right dome of the diaphragm which is at the level of the seventh rib posteriorly. The cavity previously described is no longer present. Several scars and calcified areas now occupy the area formerly occupied by the cavity. There appears to be no evidence of active pulmonary disease. For additional clinical data see chart 1.

SUMMARY

Radiographic evidence has been presented showing the actual closure of apical (fig. 5) and hilar (fig. 3) type cavities. In both these cases pneumoperitoneum was used in combination with phrenic paralysis. Artificial pneumothorax had been previously unsuccessfully attempted in both instances. Exudative type lesions have undergone various degrees of absorption and fibrosis. The degree of clearing appears to bear direct relationship to the length of time phrenic paralysis and pneumoperitoneum have been in effect. Figure 2 illustrates the amount of clearing resulting after 8 months of therapy. In this case the absorption has been quite marked, especially in the left lung fields.

In addition it is of importance to note that in most of these cases combined phrenic paralysis and pneumoperitoneum were used only after other methods had been unsuccessfully tried. In other words, pneumoperitoneum offered the only possible method of inducing pulmonary compression and immobilization.

From a clinical standpoint the results have also been promising. Chart 1 reveals the period under treatment has varied in individual cases from 3 to 9 months. The gain in weight has varied from no gain to an increase of 35 pounds. In all cases there has been a decided drop in the blood sedimentation index. The temperature range has been reduced to within normal limits. In 6 out of the 15 cases reported the sputum has become negative for *M. tuberculosis*.

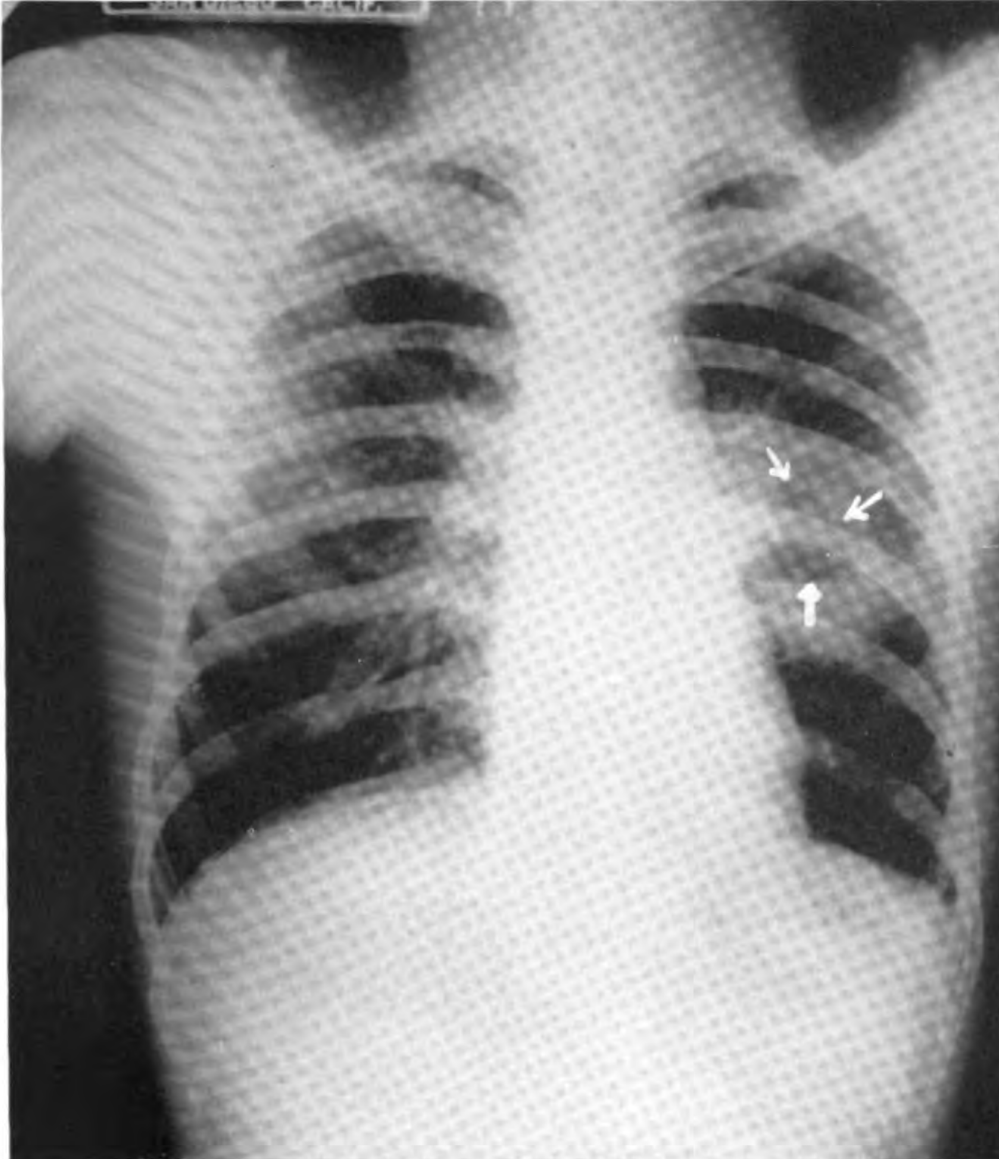


FIGURE 3.—W. F. B.—BEFORE OPERATION.

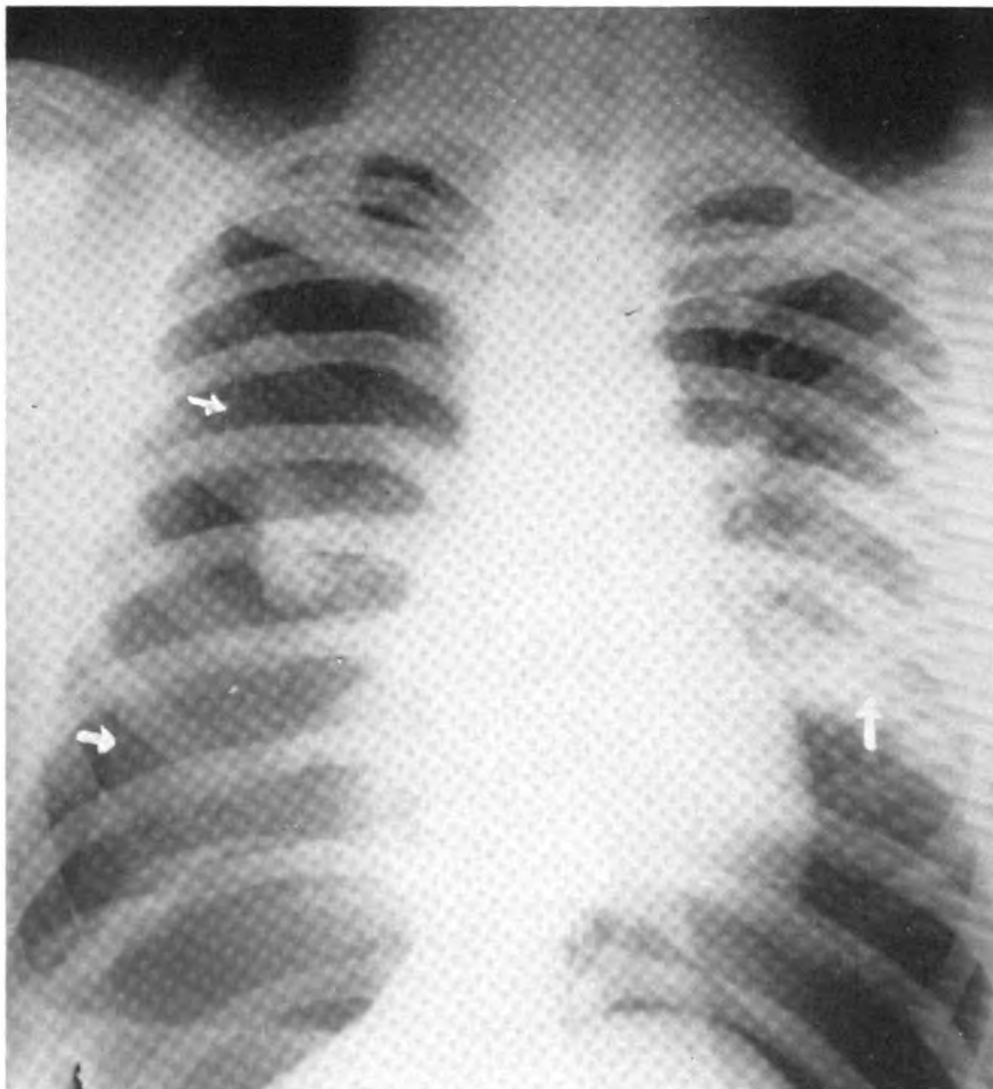


FIGURE 4.—W. F. B.—AFTER OPERATION. RIGHT, PNEUMOTHORAX. LEFT, PHRENIC PARALYSIS. PNEUMOPERITONEUM.

CHART 1

Case	Admitted	Condition on admission					Clinical history					Chest X-ray	
		Type	Weight	Sed. index	Tem-perature range	Sputum	Months hosp.	Weight gain or loss	Sed. index	Tem-perature range	Sputum		Treatment
W. F. B.	Mar. 21, 1939	Mod. adv. bilateral.	120	22	98.6 102.2	Positive	9	35+	19	98.6	Negative	Rt. pneumothorax. Left phrenic crush. Pneumoperitoneum.	Marked improvement.
F. C. C.	Aug. 29, 1939	Far adv. bilateral.	137	22	98.6 100	do	4	0	15	98.6 98.9	Positive	Bilateral phrenic crush. Pneumoperitoneum.	Improved.
T. O. D.	July 17, 1939	do	127	25	98.6 101.4	do	5	10+	21	98.6	Negative	do	Do.
N. S. F.	Sept. 8, 1939	Mod. adv. bilateral.	140	30	98.6 102.4	do	4	10+	17	98.6	do	Rt. phrenic crush. Left pneumothorax. Pneumoperitoneum.	Do.
W. M. J.	Sept. 28, 1939	Mod. adv.	140	23	98.6 102.4	do	3	12+	12	98.6	Positive	Rt. pneumothorax. Left phrenic crush. Pneumoperitoneum.	Do.
J. E. J.	do	Mod. adv. unilateral.	118	31	100 103.6	do	3	4+	25	98.6	do	Lt. phrenic crush. Pneumoperitoneum.	Do.
W. J. M.	Feb. 7, 1938	Far adv. bilateral.	125	20	98.6 99.5	do	9	10+	14	98.6	do	do	Marked improvement.
R. V.	June 2, 1939	do	143	24	98.6 102.4	do	7	10-	19	98.6	do	Rt. pneumothorax. Lt. phrenic crush. Pneumoperitoneum.	Improved.
R. T.	Apr. 27, 1939	do	145	25	98.6 100	do	9	0	15	98.6	do	do	Do.
E. Y.	Apr. 7, 1939	do	168	20	98.6 99.4	do	9	0	25	98.6	do	do	Marked improvement.
H. A. Z.	Apr. 5, 1939	do	147	25	98.6 103.6	do	8	29+	17	98.6	do	do	Improved.
C. P.	Mar. 8, 1939	Mod. adv. unilateral.	162	17	98.6 102.2	do	9	0	6	98.6	Negative	Rt. phrenic crush. Pneumoperitoneum.	Marked improvement.
G. M.	Aug. 19, 1939	Far adv. bilateral.	119	24	98.6 102.4	do	6	14+	16	98.6	do	do	Improved.
G. O.	July 3, 1939	Far adv.	127	18	98.6 100	do	7	13+	18	98.6	do	Lt. pneumothorax. Rt. phrenic crush. Pneumoperitoneum.	Do.
A. A. F.	Aug. 5, 1938	do	140	16	98.6 102	do	9	1+	12	98.6	Positive	Bilateral phrenic crush. Pneumoperitoneum.	Do.

One of the first responses to combined phrenic paralysis and pneumoperitoneum is diminished cough without decreased expectoration. The patient is enabled to keep the bronchial tract well drained without constant cough. Later as the lesion improves the amount of expectoration diminishes. Other desirable effects are improved appetite and feeling of well being. This psychic improvement is especially marked in far advanced bilateral cases in which other methods of collapse have been previously unsuccessfully attempted. The fact that something definite in the way of therapy is being instituted often changes the patient's entire mental outlook.

CONCLUSION

1. Combined pneumoperitoneum and phrenic paralysis have produced radiographic evidence of pulmonary parenchymal cavity closure and the absorption and fibrosis of exudative type lesions.
2. Clinical and symptomatic improvement has been demonstrated.
3. Combined phrenic paralysis and pneumoperitoneum have a definite place in the collapse therapy of pulmonary tuberculosis.

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STREAMLINING FULL DENTURE PROSTHESIS

By Lieutenant (Junior Grade) J. B. Casey, Dental Corps, United States Navy

In our Dental Corps, the practice of providing dental prosthesis at centrally located dental activities, rather than at each dental office has many advantages, particularly in the economical administration of the Dental Corps. However, from the viewpoint of the service as a whole, the relative inaccessibility of these centers and the consequent loss of man-hour time are disadvantages. This is especially noticeable in full denture prosthesis. Therefore a technic is desirable which would eliminate several appointments. If a denture is to be made in a few appointments, the technic must be more rigorous in order to eliminate excessive time-consuming future adjustment. The technic presented here reduces the number of appointments, gives the central-bearing-point development, and secures the centric and protrusive bite tracings. It is convenient, and provides checks on laboratory accuracy throughout.

First appointment.—Snap impressions and models are made in standard fashion. Base plates are made of black tray compound,

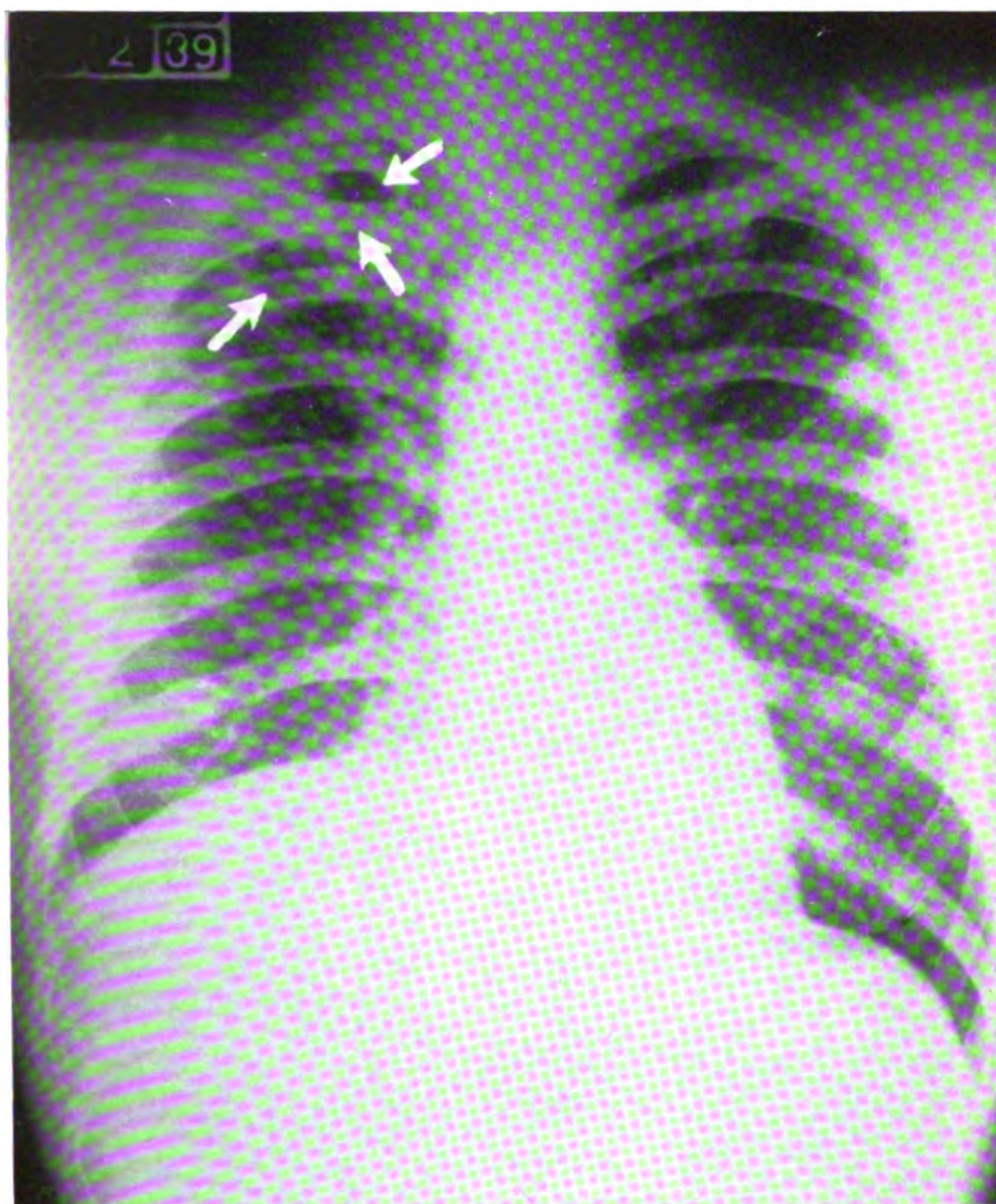


FIGURE 5.—C. P.—BEFORE OPERATION.

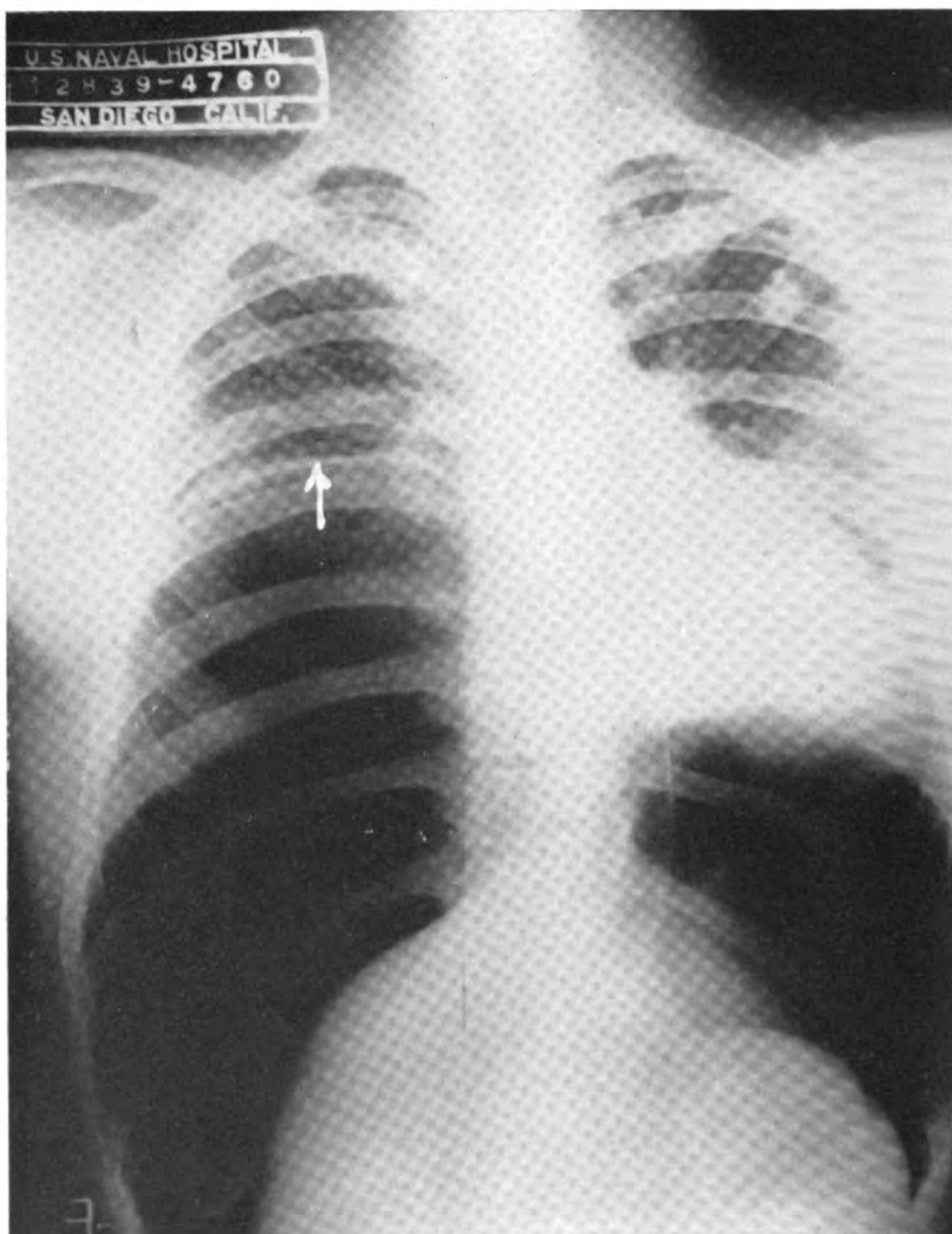


FIGURE 6.—C. P.—RIGHT, PHRENIC CRUSH. PNEUMOPERITONEUM.

not over $\frac{1}{8}$ inch thickness at heels. Narrow wax rims are adjusted, checking in the mouth as usual for height and contour.

The Warren centric instruments are inserted in the bite rims. The upper is inserted flush with the surface of the upper bite rim, and the lower instrument is sunk $\frac{1}{4}$ inch below the surface of the lower bite rim.

The central bearing point screw of the lower instrument is adjusted to just touch the plate of the upper instrument. That part of the lower rim above the lower instrument is removed flush with the lower instrument.

Notches are cut in upper and lower rims (for use with plaster bite locks). An impression corrector of the zinc-oxide-eugenol type is placed in the base plates and the final impressions made via intra-oral technic. The central-bearing-point is found very useful in this as well as in the following step.

The Gothic-arch tracings are made via the table and stylus of the Warren instruments. Protrusive and centric relations are secured with plaster bite locks. With the assembly locked in centric, the face bow assembly is adjusted and then removed from the upper instrument bearing plate and laid aside until the assembly is ready to be mounted.

The tooth shade and mould having been determined, the patient is dismissed. The final models are made, their bases cross-grooved (to aid in grind-in later), and the entire assembly mounted in the articulator, using the face bow in standard fashion. Then with the condylar inclination blocks and the incisal table of the articulator loosened so as to move freely, the protrusive bite lock is inserted in place of the centric lock, and the condylar inclination thus established. The Warren instruments are removed and the teeth set up, using the upper rim as the guide. The cases are waxed and cured in the usual fashion. The models are removed from the flasks with the plates, plaster and vulcanite removed from the model bases and the occlusal surfaces of the teeth, and the models remounted on the previously used plaster blocks in the articulator. The teeth are ground to correct occlusion in the articulator. The plates are then removed from the models and polished.

Second appointment.—The plates are inserted. The patient is told to return in 48 hours. It is believed that any adjustment at this sitting is wasted time, except in cases of recent extractions.

Third appointment.—The occlusion is checked carefully, and areas relieved as usual.

Other sittings may be arranged at the convenience of the patient for adjustments. The only sitting for which considerable time (usually about 4 hours), must be allowed is the first.

PYLEPHLEBITIS COMPLICATING APPENDICITIS

By Lieutenant Paul M. Crossland, Medical Corps, United States Navy

Pylephlebitis, or inflammation of the portal vein, is caused by an acute suppurative process adjacent to the vein, or in a part of the abdomen drained by the portal circulation. Multiple embolic abscesses of the liver always result, and consequently make pylephlebitis invariably fatal. This fact makes the prevention of this condition a most important consideration.

Numerous pathological conditions have been responsible for the development of pylephlebitis, such as acute appendicitis, infected hemorrhoids, acute suppurative cholecystitis, peridiverticular abscess, suppuration in the pelvis, and pyonephrosis with obliteration of the splenic vein. Roberts and Hadler (1) report an interesting case of pylephlebitis following a perforating ulcer on the posterior-superior wall of the duodenum. The perforation resulting in a blind fistula with its apex resting on the portal vein caused thrombophlebitis inside the venous trunk without actually perforating it.

Of all the pathological conditions resulting in pylephlebitis, acute appendicitis is the most frequent. This paper will deal principally with its development as a complication of appendicitis, and discuss its diagnosis and treatment.

INCIDENCE

As a cause of death following acute appendicitis, pylephlebitis has received very little attention. In older statistics Armstrong (2) and Petren (3) report this complication present in 5 percent of the patients dying of acute appendicitis. During the past few years the earlier operative treatment of acute appendicitis has lowered the incidence of pylephlebitis to 0.4 percent as reported by Gatch and Durman (4) and 0.3 percent by Hawkes (5).

PATHOLOGY

Busch and Spivack (6) report 4 fatal cases of pylephlebitis with liver abscess in a series of 635 cases of acute appendicitis. Two of these cases had a gangrenous appendix with perforation, the other 2 were cases of appendicial abscess. A study of other cases reported in the literature (7), (8), and (9), reveals that in each case pylephlebitis was a result of marked appendicial pathology. When such pathology is encountered at operation, one should consider the possibility of the development of this grave complication.

In cases where the organisms are particularly virulent, or the body resistance is low, or nature's effort to localize the infection have been thwarted by the use of laxatives, the organisms penetrate the local protective barriers, and quickly gain entrance into the venous radicles.

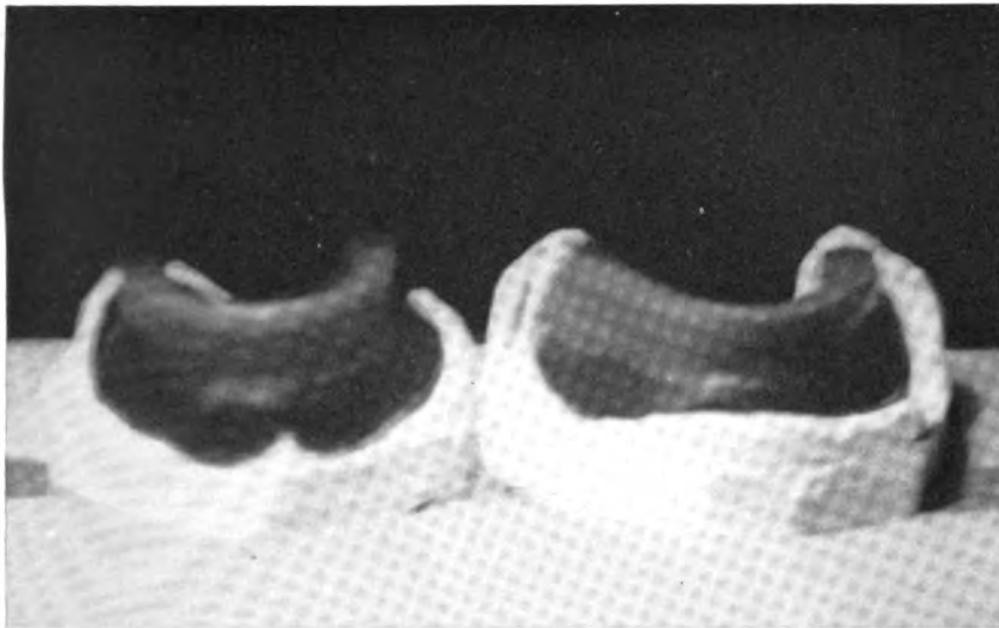


FIGURE 1.—BASE PLATES AND RIMS.



FIGURE 2.—WARREN INSTRUMENTS ATTACHED TO RIMS.

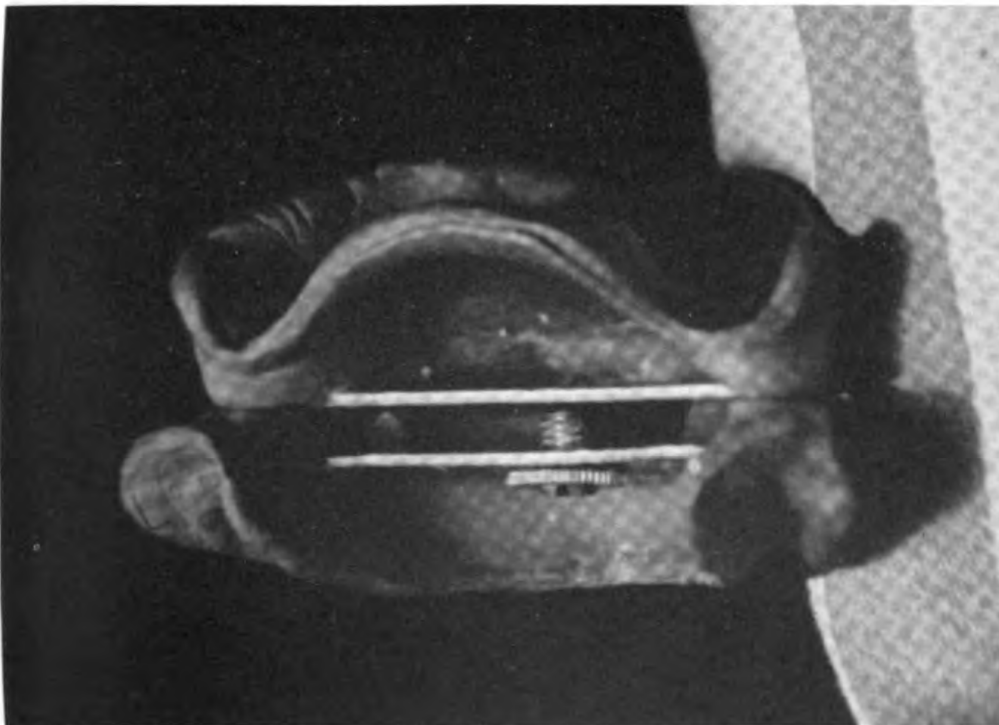


FIGURE 3.—ADJUSTMENT OF CENTRAL BEARING POINT.



FIGURE 4.—ASSEMBLY AFTER FINAL IMPRESSIONS, READY FOR RELATIONS REGISTRATION.



FIGURE 5.—ASSEMBLY LOCKED INCENTRIC AND ATTACHED TO FACE BOW.

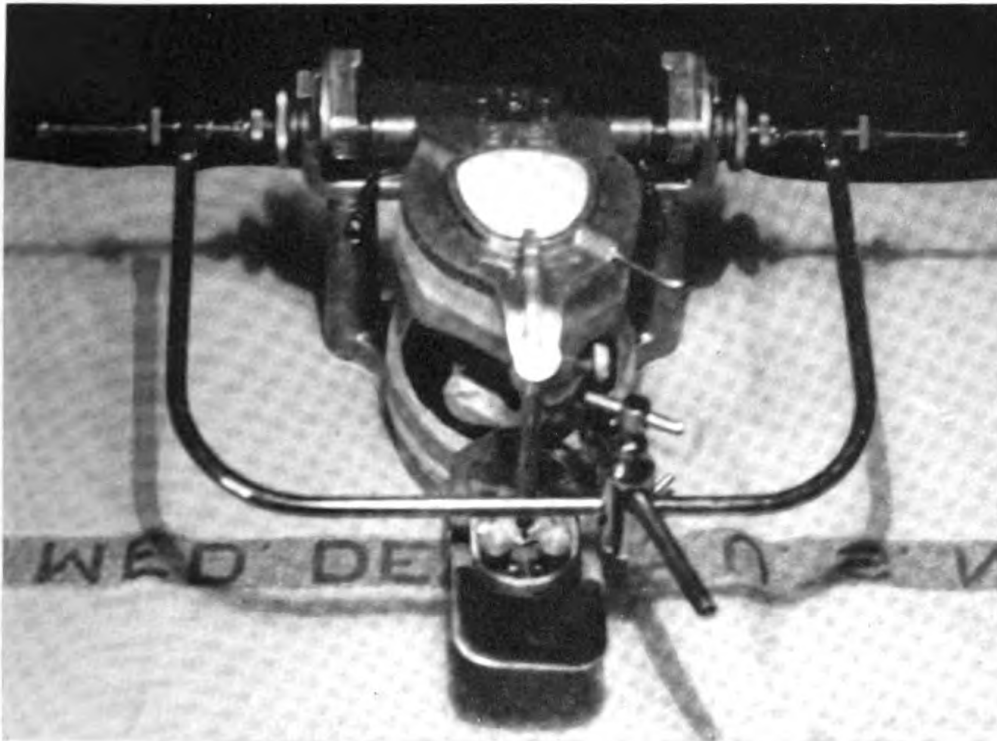


FIGURE 6.—ASSEMBLY MOUNTED IN ARTICULATOR.

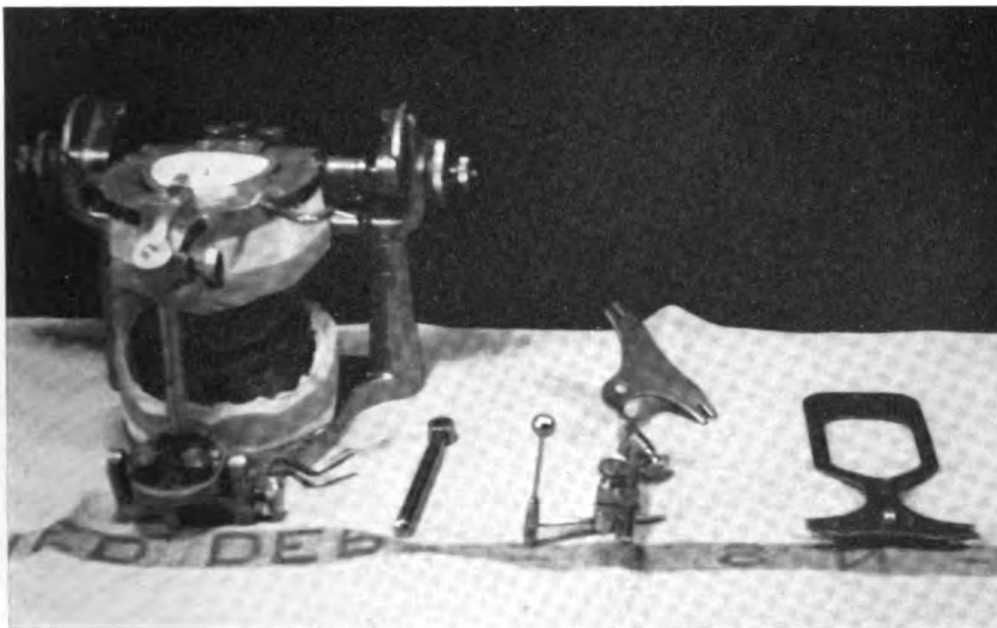


FIGURE 7.—WARREN INSTRUMENTS REMOVED AND CASE READY FOR SET-UP. THE UPPER RIM AS REGARDS HEIGHT AND CONTOUR IS CORRECT. THE LOWER, DUE TO THE CUTTING AWAY NECESSARY TO ALLOW THE CENTRAL BEARING POINT FREE MOVEMENT, IS NOT CORRECT AS A GUIDE FOR HEIGHT,

The formation of a clot in the appendicial vein results. The infected clot is an attempt to prevent the immediate spread of the infection. Progressive enlargement of the protective thrombus takes place in virulent infections and may proceed to the larger veins, or small portions of this thrombus may be detached and an infected embolus is set free in the portal circulation. In either case, its course extends from the appendicial vein to the ileo-colic vein, then to the right colic and superior mesenteric veins, or from the ileo-colic direct to the superior mesenteric, whichever the case may be. From the superior mesenteric it enters the portal vein itself and finally comes to lodge in the liver. Along the course of any of these veins small localized abscesses may form due to perivenous infection.

Jaundice may be present due to marked liver damage. Until the liver becomes riddled with embolic abscesses and its venous drainage thrombosed, the general systemic circulation may remain free of bacteria. Positive blood cultures are rare.

SYMPTOMS

In addition to the clinical history indicative of appendicitis, the patient may give a history of a chill. It may have seemed unimportant to him at the time and have to be elicited by careful questioning. It is a most significant point when it can be established that the chill was not due to some other pathological condition. His fever is usually higher and he appears more toxic than one would expect to find in the usual type of acute inflammation of the appendix.

In the postoperative course of a gangrenous appendix or an appendicial abscess, the occurrence of one or more distinct chills, accompanied by fever and prostration, are important diagnostic signs. They usually indicate dissemination of the pyemic process with probable development of metastatic abscesses.

Slight jaundice may develop and the liver may be enlarged and tender. Abdominal pain usually is not significant; however, it may be severe and more or less generalized.

DIFFERENTIAL DIAGNOSIS

There are several conditions which should be ruled out before a diagnosis of pylephlebitis complicating appendicitis can be made. Right-sided pyelitis may give a similar picture of chills and fever; however, tenderness is located more in the right flank and the signs of peritoneal irritation are usually absent. Examination of the urine is most important, and will usually show evidence of urinary infection.

Early pneumonia may cause confusion but can be ruled out by careful physical examination and bedside x-rays.

When signs have shifted to the upper right quadrant, subdiaphragmatic abscess and gall bladder disease must be considered. Right sided pelvic inflammatory disease and early deep phlebitis of the right lower extremity may be confusing.

The diagnosis must be made early before irreparable damage has taken place. It is possible that the liver can overcome a small dose of infected emboli but repeated large doses are certain to cause death.

TREATMENT

The best treatment of pylephlebitis is prophylaxis, that is, early operation before the development of gangrene, or an infected thrombus in the appendicial vein. If all patients were operated on within 24 hours, this complication would not be encountered.

Treatment of pylephlebitis, per se, by operative intervention, has been most disappointing. It has consisted chiefly in attempts to keep the progressive thrombus or infected emboli from reaching the portal vein.

Wilms (10) was the first to suggest ligation of the veins of the ileo-colic angle and in one case successfully prevented the extension of the thrombotic process. Later Braun (11) recommended the ligation of the ileo-colic vein at its junction with the superior mesenteric because necropsy in four fatal cases showed that the thrombus had not extended beyond the ileo-colic vein. He performed this operation successfully in two cases. More recently Hawkes (5), in several of his cases when the thrombus was limited to one small radicle of the appendicial vein, dissected well behind the ileo-cecal junction and ligated the appendicial vein high above the thrombus.

Routine ligation of the ileo-colic vein in every case of acute appendicitis is not recommended. However, the meso-appendix should always be inspected for thrombosed vessels and, if found, ligated well above the thrombus. If there is a history of one or more chills, or in late cases, with fever of the remittent (spiking) type, the ileo-colic vein should be ligated. In the post-operative course the occurrence of a distinct chill followed by fever and prostration, other complications being eliminated, a second operation of ligation of the ileo-colic vein should be performed immediately.

The right rectus incision with its upper end opposite the umbilicus is the incision of choice for giving the maximum exposure. By placing the patient slightly on the left side the small intestine tends to fall away from the right gutter. After they are packed away with warm sponges a clear view of the ileo-cecal junction is obtained.

The small tributaries of the ileo-cecal vein can be seen beginning to form a common trunk which runs in an oblique line from the

ileo-cecal angle toward a point midway between the umbilicus and the xiphoid process. It will be found to join the right colic vein just below the third portion of the duodenum. Occasionally the ileo-colic empties directly into the superior mesenteric below the right colic vein. Lying to its medial side throughout its course is the ileo-colic artery. The vein thus identified is gently palpated for the presence of a thrombus which, if present, stands out as a stiff cord-like structure. An attempt should be made to ligate the vein at a point above the thrombus. Even though no thrombus is found the ileo-colic vein should be isolated, doubly ligated as close as possible to its junction with the right colic vein, and severed. This procedure will prevent infected emboli from entering the larger veins of the portal circulation and the formation of liver abscesses.

There need be no fear of causing gangrene by this procedure as the right colic and lower branches of the superior mesenteric veins anastomose with the ileo-colic vein and aid in draining this area. Stewart-Wallace (8) reports the ligation of the superior mesenteric vein within an inch of its junction with the splenic vein in a case of pylephlebitis following appendicitis. Ligation was followed by a sudden and marked engorgement of all the colic veins and shock. However, the patient responded to treatment and recovered completely. Colp (12) cites three cases of ligation of the portal vein without ensuing gangrene; however his patients died later because of liver abscesses.

Sulfanilamide has been used in the treatment of pylephlebitis with liver abscess. Ottenberg and Berck (9) report two cases, one from an infected hemorrhoid, the other from a gangrenous appendix. In neither case was vein ligation above the infected thrombus done. Both cases completely recovered.

It is doubtful that surgical intervention can accomplish anything once the thrombotic process has passed beyond the ileo-colic vein. Our only hope in the surgical treatment of pylephlebitis is to prevent its development by keeping the process limited to this vein.

CASE REPORT

R. T. J.—Veterans' Administration patient, age 51, admitted on January 30, 1940, ambulant, complaining of pain in right lower abdomen. Two days previously he had generalized abdominal pain, nausea and vomiting, followed by localization of pain in the right lower quadrant. He had taken epsom salts early in the attack and an enema the night before admission. A large hernia in the right kidney region following a nephropexy 4 years previously had confused the first attending physician and delayed the diagnosis.

The patient appeared apprehensive and acutely ill. Temperature 99.6°, pulse 130, respirations 22. There was marked tenderness and rebound tenderness just medial to the right anterior superior spine. No mass was palpable. In the right loin was a large postoperative hernia easily reducible but not tender. His

abdomen was opened through a McBurney incision. A ruptured gangrenous appendix was found plastered to the parietal peritoneum of the flank with early abscess formation. There was early gangrene of the posterior parietal peritoneum extending into the retroperitoneal tissues. Appendectomy with drainage was performed.

After operation, treatment for peritonitis was instituted, consisting of continuous intravenous drip, morphine, and Wangensteen suction siphonage.

On the second postoperative day the patient had a distinct chill lasting 20 minutes, followed by a temperature of 103°. He did not complain of any abdominal pain.

On the third postoperative day a mild phlebitis was present in the saphenous vein of the left leg which had been used for the intravenous drip. He had a second chill with fever similar to the one the day before.

He continued to have a daily chill, each more severe than the day before, followed by a higher fever. By the tenth postoperative day the chills were lasting 30 to 40 minutes followed by fever between 105° and 106°. The phlebitis in the left leg had practically subsided. He had complained of very little abdominal pain and tenderness. Distention had been moderate. It was noted that he was mildly jaundiced. His icterus index was 24. Blood urea nitrogen and blood creatinin only slightly elevated. Blood culture showed staphylococcus aureus. Physical examination and x-rays of chest and abdomen were negative for lung abscesses, pneumonia, or subdiaphragmatic abscess. Liver was not enlarged.

During the next 15 days he continued to have at least one and sometimes two chills each day, followed by high fever for a few hours. Sulfanilamide was given in large doses without any appreciable effect on the chills or fever. Three blood transfusions made the patient feel stronger, but had no other effect. Bacteriophage made from his positive blood culture seemed to be of some benefit as his chills were not as severe and his fever did not go as high as before this treatment was started.

However, on the twenty-fifth postoperative day he became markedly distended and irrational at times. Treatment was of no avail. He became comatose and died on his twenty-seventh postoperative day.

Autopsy revealed thrombosis of the small mesenteric veins. The mesentery of the ileum measured 1½ cm. in thickness, was indurated, and on section contained numerous small abscesses. The larger mesenteric veins and portal vein contained many necrotic mural thrombi and numerous small abscesses were found along the route of these vessels. In the liver were many abscesses containing necrotic thrombi. There was a localized abscess in the lesser peritoneal cavity and another among the loops of small bowel in the right lower quadrant. The lungs, heart, spleen, and kidneys were not involved.

SUMMARY

1. Pylephlebitis following acute appendicitis is an infrequent but grave complication, and is a result of marked appendicial pathology.
2. A thrombosis of the appendicial vein may progressively enlarge and extend to the larger vessels of the portal circulation, or infected emboli may be set free resulting in liver abscesses.
3. Chills, fever, and prostration are important diagnostic signs and indicate extension or dissemination of the pyemic process.
4. The best treatment is early operation for appendicitis. Ligation of the ileo-colic vein is indicated if there are chills, fever, and prostra-

tion, either preoperatively or postoperatively in a case of acute appendicitis.

5. A fatal case of pylephlebitis complicating appendicitis is reported.

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NEURITIS AS A SERUM REACTION

REPORT OF TWO CASES

By Lieutenant Commander B. W. Hogan, Medical Corps, United States Navy

It is only in recent years that neuritis following serum injections was recognized. Lhermitte (France) first described these nervous disorders in 1919. According to A. E. Bennett (1) the literature up to 1938 revealed about 70 French cases, 29 American and English cases, 10 German and other cases, totaling 115. Serum polyneuritis is most often seen after the administration of antitetanus serum, probably because this serum is more frequently used. The next most common cause of neuritis from the use of a foreign protein is the pneumococcus serum. There are also reactions occurring after the use of scarlet fever, meningococcus, streptococcus, and diphtheria sera. In fact all foreign proteins cause a reaction.

The etiology is unknown. Serum sickness is most common and severe after the use of fresh serum rather than after old preparations. The serum from some horses causes greater reactions than from others. Other partial factors are age, serum dosage, purification, concentration, and route of administration.

The disorder occurs predominantly in persons over 21 years of age. Males are more often affected than females, probably owing to the relatively greater frequency of accidents among males, with the consequent more frequent use of antitetanus serum. There is no adequate explanation of the relative immunity to neurologic disorders of most patients who receive serum.

Bennett (1), suggests four theories for the pathogenesis of serum neuritis:

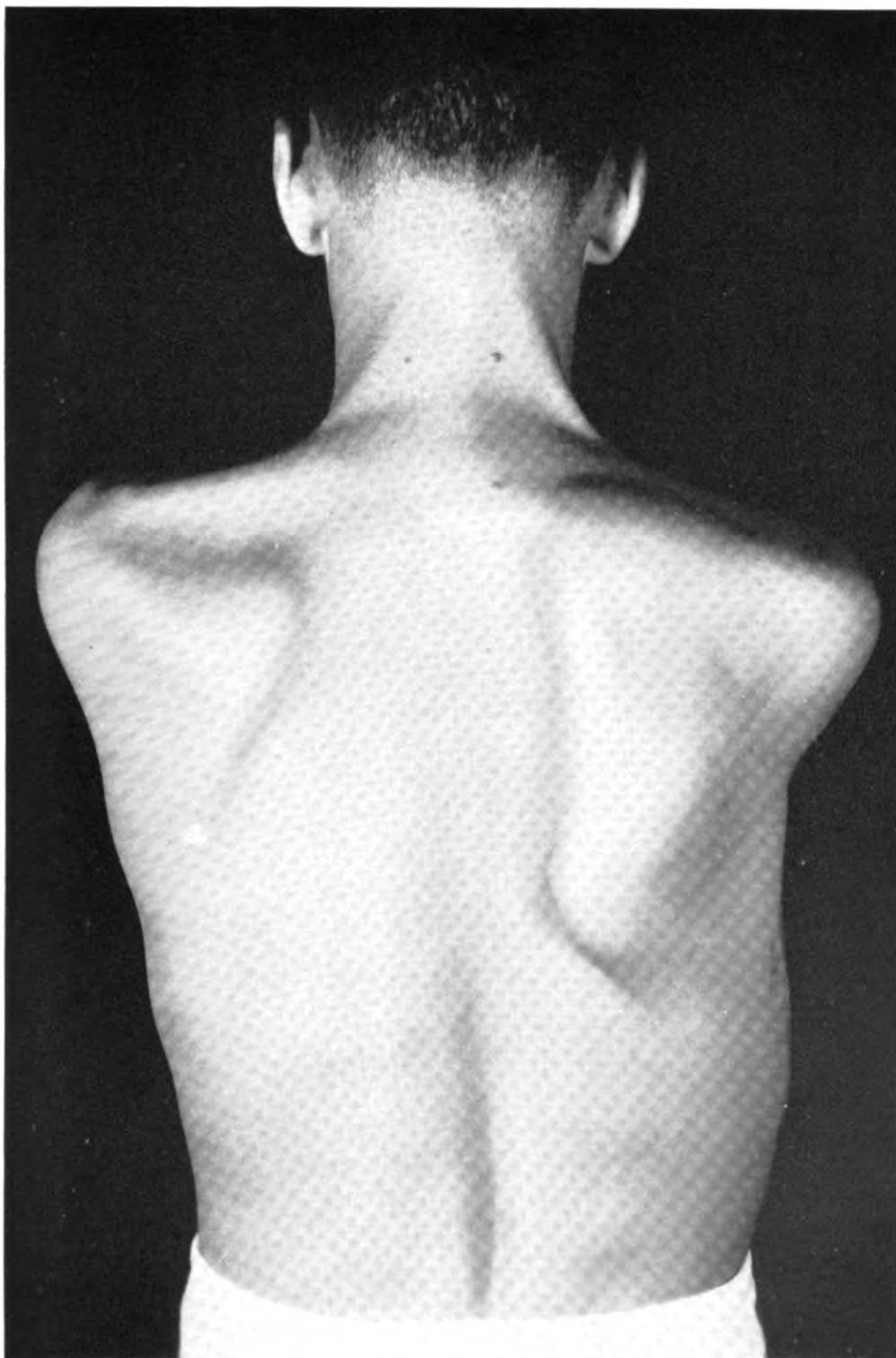
1. Direct toxic action of serum;
2. Selective action of toxins on nerves with low chronaxias;
3. Compression from perineural edema of sheaths about the intervertebral foramina or bony grooves; and
4. Vascular origin, nerve-cell death from vasodilation, perivascular infiltration and hemorrhage.

Kraus and Chaney (2) write concerning the pathologic process: "It seems that the neuropathologic changes associated with serum sickness are the same as those which appear elsewhere in the body and consist of a primary disorder of the blood vessels, causing nutritive impairment of the tissues of the nervous system interfering temporarily as a rule with the activity of the nerve fibers and cells but occasionally causing cell death and parenchymal necrosis. A vascular disorder also produces the meningeal picture and excess spinal fluid. An edematous process may occur in the perineural sheaths, or the nutrition of the roots or nerves may be interfered with by impairment in the blood supply, thus causing various radicular and neural syndromes."

It is this writer's feeling that a perineural swelling causes a temporary physiological block of nerve function or compression neuritis.

CLINICAL VARIETIES

The various clinical manifestations seen depend upon the nervous tissue affected. There may be meningeal signs and symptoms or evidence of cerebral or spinal cord lesions. However, the most common reactions occur in the peripheral nerves, usually affecting the upper brachial plexus and the roots of the fifth and sixth cervical nerves either unilateral or bilaterally. In the great majority of cases, the nerves supplying the shoulder muscles are involved. Infrequently the cranial nerves are involved and in exceedingly rare



CASE II.—R. F. E.—PARALYSIS OF RIGHT SERRATUS MAGNUS, GIVING WINGED SCAPULA. ATROPHY OF SHOULDER MUSCLES. BULGING RIGHT CHEST WALL.

cases, the nerves supplying the lower limbs, which give rise to muscular and sensory disturbances of the lower extremities.

The serious clinical entity, known as acute ascending paralysis or Landry's paralysis, has been reported as following antitetanus serum inoculation.

The usual clinical picture is that of a person who is injured and is given tetanus antitoxin or who suffers from one of the infections previously mentioned. He is given therapeutic serum, and suddenly shows new symptoms about a week after receiving it. There may be fever and urticaria, either generalized or limited to the region of the injection, joint swelling, enlarged and painful lymph nodes and headache.

The symptoms of neuritis occur at the height of the serum sickness, with severe pains in the neck, shoulders, arms or legs, unrelieved by analgesics. Within hours to a few days, flaccid paralysis develops, followed by muscular atrophy with muscular tenderness and dull pains persisting for weeks. Reduced reflexes, anesthesia, motor paralysis, and at times fibrillations occur, depending on the segments involved.

The duration of this illness varies. In some cases weakness and paralysis may persist for 4 weeks or for 1½ years. About 20 percent are left with some residual atrophy and weakness, usually in the deltoid muscles.

THERAPY

The treatment consists in:

1. Early recognition.
2. Protection of involved limbs.
3. Adrenalin, dehydration, and fever therapy.
4. Vitamine therapy.
5. Local heat and analgesics.
6. Physiotherapy, massage, and electrical stimulation.

PROGNOSIS

The prognosis is good. Eighty percent recover entirely. Twenty percent have a residual atrophy with little incapacity.

CASE REPORTS

Case I.—R. A. H., coxswain, United States Navy, age 27, married, admitted to the hospital ship *Relief* December 1, 1939. Temperature, 99°; pulse, 80; respirations, 18. Complained of "great difficulty in walking and pain in calf muscles."

HISTORY: The history revealed that after he was injured by wood splinters piercing his right index finger, November 20, he received 1,500 units of tetanus antitoxin into the left buttock. Preliminary sensitivity tests showed no reac-

tion. November 23, he was nauseated and feverish; November 24, he had chills and fever; November 26, he had generalized urticaria, itching, and fever; November 27, frequency in urination, very painful sensation in calves of legs, difficulty in walking; November 28, walking reduced to a shuffle, he had to sit down every few minutes. Pain in lower limbs was severe. He had to pull himself up the ladder by the use of his hands and arms. Bladder symptoms of frequency and urgency continued.

EXAMINATION: Neurological examination on admission showed that he was unable to walk or stand on his toes and there was difficulty in walking on heels. Shuffled when walking on feet. Gait was unsteady due to weakness. No tremors. No fibrillations. Motor weakness in all movements of legs and feet. No atrophy. Cranial nerves intact. Reflexes—superficial present, equal and active. Deep reflexes of the upper extremities hyperactive but equal; lower extremities, knee kicks hyperactive and equal and ankle jerks sluggish to absent. No clonus. Babinski negative. Sense of position disturbed in right large toe otherwise normal. Vibration sense normal. Sensory—there was a blunting in recognition of pain, temperature and light touch in the distribution of the common peroneal and posterior tibial nerve. Pain in the gastrocnemius and soleus muscles was fairly severe and there was marked tenderness when pressure was applied to these muscles. No trophic or vaso-motor disturbances.

LABORATORY FINDINGS: Blood Kahn negative. Urinalysis normal. Red blood cells, 5,040,000. White blood cells, 7,100. Haemoglobin 14 grams. Differential: Bands, 4; segs, 49; lymph, 29; eosino, 7; baso, 3; mono, 8.

Spinal puncture: Fluid clear. Pressure 110 mm. of water with bilateral jugular pressure rise and quick fall. Cell count 5. Globulin, no increase. Protein estimation 25 mgm. per 100 cc. Kahn negative. Gold curve 0000000000. Blood sedimentation rate 2 mm. per 60 minutes. Blood chemistry: Urea nitrogen 11 mgm. per 100 cc., blood sugar 90 mgm. per 100 cc., and blood chlorides 490 mgm. per 100 cc.

TREATMENT: Absolute bed rest. Support and heat to lower extremities. High vitamin B-1 and B-2 intake. Physiotherapy and swimming during late convalescence.

PROGRESS: Pains and soreness subsided in 7 days. Patellar reflexes became sluggish and ankle jerks remained sluggish or absent. Strength returned after 2 weeks and full use of legs in 4 weeks. No residuals found.

Case II.—R. F. E. Ph. M. Sc., 26 years old, single, admitted May 28, 1940.

He had been bitten on the index finger of right hand on May 18 and was given 1,500 units of tetanus antitoxin in the left lower deltoid area. The sensitivity tests showed no reaction. May 22, there was redness and itching around the area of previous injection. May 24, generalized urticaria, crops of small and large wheals. May 25, severe pains in distribution of fourth and fifth cervical root areas. Pain increased on moving of arms. May 27, fell to deck while taking a shower, experienced paresthesias, numbness, heaviness, cold and stinging sensations radiating up from feet to mid-thigh, then loss of strength in legs. For the next 2 or 3 days he was unable to stand on legs longer than 45 to 90 seconds. He was unable to raise arms to or above shoulder level. In the previous months patient had suffered from frequent upper respiratory infections.

EXAMINATION: Temperature 98.6°, pulse 92, respirations 20. Blood pressure 110/70. Head and neck, heart and lungs, abdomen and genitalia negative.

Neurological: Gait normal. Romberg negative. Coordination normal. Cranial nerves intact. No tremors. No fibrillations. Sensory tests normal throughout except for blunting of light touch over the fifth cervical distribution bilaterally. Vibration, sense of position and sphincters intact. Upper arms felt

cold, forearms warm. Poor muscle tone in shoulder area and all extremities. More marked in upper limbs. Hand grips poor; unable to raise left arm to shoulder level or above. Unable to raise right arm above shoulder level. Abduction and adduction movements of arms diminished. Winged scapula on right side. Right buttock lags and hangs lower than left. Bulging of the lower part of right thoracic wall. Reflexes: Superficial—left cremasterics and abdominals not as active as right side. Deep—right triceps absent; left present. Right biceps sluggish; left shows slight reaction. Knee kicks more active on right than left. Ankle jerks more active on right than left. Babinski negative. No clonus.

LABORATORY FINDINGS: Blood Kahn negative. Blood sedimentation rate 10 mm. per 60 minutes. Blood chemistry: Blood chlorides 518 mgm. per 100 cc., urea nitrogen 12 mgm. per 100 cc., and blood sugar 58 mgm. per 100 cc. Red blood cells, 4,810,000. White blood cells, 8,050. Haemoglobin 84 percent. Differential: Bands, 4; segs, 74; lymph, 15; eosino, 4; mono, 3. Urinalysis negative.

Spinal puncture on admission, May 28, 1940: Clear fluid. Normal pressure. No evidence of block. Cell count 50 (lymphocytes). Total protein 40 mgm. per 100 cc. Globulin slight increase. Kahn negative. Gold curve 0000000000. June 11, 1940: Spinal puncture. Cells 8. Total proteins 30 mgm. per 100 cc. Chlorides 793 mgm. per 100 cc. Sugar 57 mgm. per 100 cc. Kahn negative. Gold curve negative.

THERAPY: Bed rest, heat, support, analgesics, forced spinal fluid drainage. Physiotherapy and swimming. High vitamin B intake.

PROGRESS: Patient remained on the sick list for 2½ months. During the first few weeks the pain in posterior neck and over shoulders extending into arms was severe and not relieved by analgesics. After 4 weeks the acute pains subsided. There was noticeable atrophy of the deltoids and shoulder muscles. Weakness in legs persisted for a month.

The muscle tone gradually increased and he was discharged August 13, 1940, with few residuals. A paralysis of the serratus magnus muscle supplied by the long thoracic nerve gave a pronounced winged scapula, the principal resulting disability. The photograph illustrates this deformity.

COMMENT

Two cases of peripheral neuritis were described. One could be classed as a neuronitis, following the injection of 1,500 units of anti-tetanus serum.

This distinct clinical entity, a complication which may follow the use of serotherapy, should be more widely recognized.

It is important to know that it has a favorable prognosis.

As most cases develop from industrial or highway injuries requiring prophylactic tetanus antitoxin, they present economic, medico-legal and compensation problems especially amongst the class of patients that the Navy medical officer treats in navy yard or out-patient duty.

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ACUTE CADMIUM METAL POISONING

REPORT OF THREE OUTBREAKS

By Lieutenant (Junior Grade) J. T. Cangelosi, Medical Corps, United States Navy

Cadmium, a toxic metal, has been hitherto little known as a cause of food poisoning. Due to its use as metal plating in a certain type of food container extensively used in the Fleet Marine Force to serve men in the field, several large outbreaks of poisoning have occurred in recent months.

Fifty-three cases of poisoning occurred at Culebra, P. R., on February 13, 1941, during a landing operation by Marines from the transport U. S. S. *Harry Lee*. Troops from combat team No. 2 were landed on Culebra Island on February 12, 1941, and remained ashore overnight. Early the next morning, 53 acutely ill patients were sent back to the ship complaining of abdominal pain, crampy in character and of sudden onset, accompanied by nausea and vomiting. Mild diarrhea occurred in approximately half of the cases. Normal or subnormal temperatures accompanied by sweating and headache was characteristic of the condition. All had mild prostration but in four cases it was moderately severe. The severity of the nausea and vomiting was out of proportion to the amount of diarrhea. There was an undetermined number who suffered from mild symptoms of vague abdominal discomforts and nausea, who did not report to the sick bay. White blood and Schilling differential counts performed on 3 cases were within normal limits. The urinalyses were negative.

The symptoms lasted 1 or 2 hours and recovery was complete in every case approximately 6 hours after the appearance of symptoms. Symptoms were transitory so every case was discharged from the sick bay on the same day. No treatment other than administering warm water by mouth to aid gastric lavage was given.

The onset of the symptoms occurred 25 to 45 minutes after the drinking of hot coffee which had been brought ashore in cadmium plated aluminum food containers. Ten men had only coffee for breakfast and these seemed to be the most severely affected. There were no cases among those who had not drunk coffee. The very short time interval between drinking of the coffee and the almost immediate sudden onset of symptoms, plus the fact that the severity of the nausea and vomiting were out of all proportion to the diarrhea which was mild, seemed to signify a heavy metal poisoning rather

than a bacterial food poisoning. The short duration of the symptoms, rapid recovery, and lack of fever were also noteworthy.

Two other outbreaks of poisoning of similar nature have occurred. On October 20, 1940, 31 men of the First Marine Aircraft Group, Fleet Marine Force, stationed at Guantanamo Bay, Cuba, reported to the sick bay complaining of vomiting, abdominal cramps, and diarrhea. The onset was sudden and occurred within 2 hours after the noon meal. Diarrhea was mild and vomiting violent. Recovery from the acute symptoms was reported within 4 hours after the onset. There were no complications. All of these cases drank lemonade at the noon meal which had been prepared in the cadmium plated food containers $1\frac{1}{2}$ hours before consumption. The ice was from Government inspected and approved sources and was used in another mess without ill effects. The food served at this mess had previously been properly prepared and served, thus eliminating food poisoning.

On February 6, 1941, 79 men from the Second Battalion, Seventh Marines, stationed at Guantanamo Bay, Cuba, reported to the sick bay with symptoms of nausea, vomiting, abdominal cramps, and diarrhea. The onset occurred 30 minutes to 5 hours following the noon meal. In about one-third of the cases the symptoms were very mild consisting only of mild abdominal discomfort. The temperature was normal or subnormal in all but 2 cases, 1 of whom had a temperature of 100° F. and the other 102° F.

All of the affected cases drank lemonade which was served in the field from cadmium plated containers. The rest of the battalion mess, comprising 641 men, had eaten the same menu for the previous 48 hours with no ill effects. Preparation and serving was proper and eliminated bacterial contamination of the food as a possible cause. Fifty-six of the affected cases were discharged to duty the day after the outbreak and the remaining 23 had been returned to duty by the morning of the fifth day.

COMMENT

All three of these outbreaks of poisoning have certain characteristics in common. In the first instance, the serving of coffee and in the other two instances, the serving of lemonade in cadmium plated metal containers caused the outbreaks. These beverages had been in contact with the metal for periods of $1\frac{1}{2}$ to 14 hours. The time interval between the drinking of the coffee or lemonade and the appearance of the symptoms of poisoning was very short. It varied from 25 minutes to 5 hours, but in the majority of cases, it was less than 2 hours. This interval seems to have been affected by the amount

of food taken with the poisoned beverage, which apparently delayed the appearance of symptoms.

The symptoms were all similar and consisted of vomiting and colicky abdominal pains of sudden onset. The diarrhea was in most instances mild, and the temperature rose above normal in only two cases. Prostration was generally mild but severe in some cases.

Recovery was rapid, and all but 23 cases were returned to duty within 1 day. The acute symptoms did not last more than 4 hours. The remaining 23 cases were retained on the sick list for 4 days. Apparently, the severity of the symptoms was influenced by the quantity of the food taken with the beverage as demonstrated by the fact that the sickest cases were generally those who had eaten the least food. There were no residual signs or symptoms in any cases.

Treatment consisted in the administration of draughts of warm water to encourage emesis. Some of the cases were given bismuth subnitrate and paregoric to control the diarrhea, but emesis and purgation were encouraged so that the metal could be eliminated as quickly as possible.

SUMMARY AND CONCLUSION

1. Two hundred and eight cases of acute metallic food poisoning in the Fleet Marine Force caused by cadmium have been reported.

2. All cases were characterized by brief duration of symptoms and rapid recovery.

3. These outbreaks have been reported to the manufacturers who are plainly marking these containers to prevent future accidental poisonings.

Note.—Specimens of lemonade involved in the outbreak which occurred in the Second Battalion, Seventh Marines, were submitted to the U. S. Naval Medical School by Lieutenant William New, Medical Corps, United States Navy, with the following results: Bacteriologically the findings were negative after incubation for 72 hours. Quantitative analysis revealed 10 mg of cadmium per 100 cc of lemonade (100 parts per million). A sample of fresh lemonade which had been left experimentally in the food carrier for 24 hours was found to contain 25 mg of cadmium per 100 cc (250 parts per million). Fifteen parts per million have been found capable of causing mild toxic symptoms.

CLINICAL NOTES

PSORIASIS NUMMULARIS

CASE REPORT

By Lieutenant Commander David B. Peters, Medical Corps, United States Navy

In view of the comparative rarity of this skin condition in the Navy, it is felt that this case is of special interest.

Psoriasis is a chronic, occasionally acute, inflammatory disease characterized by reddish-brown, flat papules, or circumscribed plaques or areas of varying sizes, covered with silvery white imbricated scales.

A ship's cook, first-class, was ordered to active duty and on physical examination, a middle-aged man was seen. His hair was quite gray and he was obese, weighing 230 pounds. The skin was pasty but the thyroid gland was of normal size; blood pressure was 136 over 78; the heart and lungs were normal; Kahn was negative. The skin over the abdomen, chest and back showed generalized psoriasis, with a few annular lesions on chest of a bright red color. The lesions, though numerous, were limited to the sweat ducts (*psoriasis punctata*). The scales were abundant and thickly concentrated over small areas of the back and abdomen. They were almost free around the waistline due to the friction of clothing and perspiration. Where the epidermis was thin, such as the inner surfaces of the arms and legs, the scalings were less. On the back of the hands the scalings were less than over the flexor surfaces. There were no glandular enlargements and the nervous system was apparently normal. The blood and urine were negative.

This man served 16 years in the Navy and upon being retired from active duty he was transferred to the Fleet Reserve. His subsequent occupation as a farmer aggravated his condition.

Family history.—Essentially negative.

Personal history.—Enlisted in the Navy on June 6, 1922. About 3 years after his enlistment a few red patches on the elbow of his left arm were noticed. Local applications of bland lotions improved the condition so that it became less noticeable. During his second enlistment, he gained more weight and a few spots appeared on his chest and abdomen which disappeared after taking Fowler's solution, 2 drops tid, and local applications of zinc oxide ointment. During

his fourth enlistment, he was transferred to the Orient, where climatic conditions aggravated his condition to the extent that all of the abdomen and back were covered with spots. X-ray treatment together with Fowler's solution soon improved this condition. He was returned to the mainland and in 1938 was released from active duty, when he returned to his farm.



FIGURE 1.—ILLUSTRATION SHOWING TYPICAL LESIONS OF PSORIASIS NUMMULARIS.

MEDICAL AND SURGICAL DEVICES

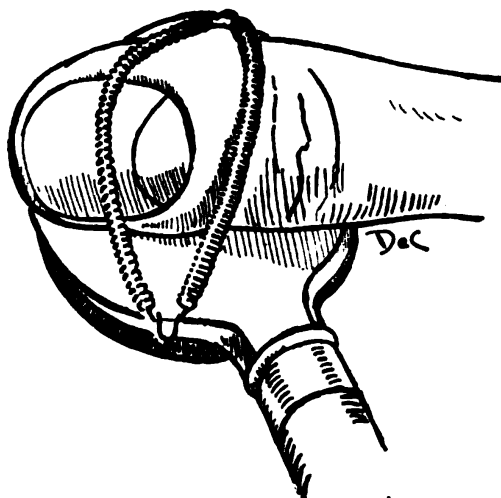
A MODIFICATION OF THE DIAPHRAGM STETHOSCOPE

By Lieutenant Edwin J. DeCosta, Medical Corps, United States Naval Reserve

With so many physical examinations being done on men inducted into the military service, any device that will facilitate physical examinations should be particularly useful. For those who are partial to the diaphragm type of stethoscope, the simple modification illustrated is proposed to render certain styles of this instrument more serviceable.

Two metal loops or "ears" are soldered, one on either side of the chest piece, at about a 45° angle from the line of the tubing. Either an elastic band or coiled metal spring is hooked over the "ears." A finger, usually the thumb, is then inserted between the elastic and the case, giving a good hold on the diaphragm with but one finger. At the same time, the tubing will be out of the way whether the patient is in the standing or recumbent position. The diaphragm can be readily moved from place to place with minimal effort and strain, and cannot slip from one's grasp.

Experience with this modified chest piece indicates that it has distinct value.



STETHOSCOPE HOLDER.

A WALKING APPLIANCE FOR PLASTER CASTS

By Blake E. Ferrell, Machinist Mate, First Class, United States Navy

Introduction.—While wearing a plaster cast with walking iron attachment for the treatment of an injured ankle in April 1940, the author felt that there was a definite need for a device which would give stabilization and absorb shocks in walking on irregular and

hard surfaces. The apparatus presented and illustrated was designed to fulfill these requirements, and in a recent limited clinical trial at the Station Dispensary, United States Naval Station, Guantanamo Bay, Cuba, has proven useful.

Description.—The walking appliance described is designed for use on nonpadded plaster casts of the lower extremity and embodies a new application of the principle of “knee-action” springs used in automobiles.

Its construction, in reference to the accompanying illustrations, is described as follows:

FIGURE A-1: Complete assembly of weight bearing end of walking appliance showing corrugated rubber cap.

FIGURE 1: Machined cap 1½-inch diameter, SAE thread. ⅜-inch threads from top to bottom. Drill ⅜-inch hole for SAE thread for attaching figure 6.

FIGURE 2: Cylinder 1 inch inside diameter, 1⅜ inches outside diameter. Thread 1⅜ inch SAE for cap (figure 1). Drill ½-inch hole in center of cylinder for plunger rod (figure 4). Cylinder 1½ inches deep.

FIGURE 3: Steel spring, 4 coil, helix type, full tension 125 pounds.

FIGURE 4: Plunger—piston end, 0.0998 inch diameter; rod, 0.0498 inch in diameter; piston thickness ¼ inch; shaft 1⅜ inches long; thread ½ inch SAE for attaching figure 5.

FIGURE 5: Lock, nut, round type, 1 inch diameter. Drill ½-inch hole in center, thread ½ inch, SAE thickness ¼ inch; drill ⅛-inch hole for Allen setscrew. Figure 5 is also used as lock nut where figure 1 and figure 6 are attached and ⅛-inch hole for padeye to act as setscrew; also for securing the two walking irons together in the cast. Modified for securing padeye, can be used in cylinder wall by drilling ⅛-inch hole and thread. The first method is advised to maintain lubrication in the cylinder wall.

FIGURE 6: The shaft made of ⅜-inch round stock, ⅜-inch SAE thread. One full inch of threads; ½ inch from threads heat and flatten to ⅝ inch in width, ⅛ inch thick, 6 inches long. From strap to bottom of threads to be 12 inches.

A photograph of the walking appliance assembled and applied to cast is presented. Note that the distal end of shaft is a separately attached piece and not a continuation of the shaft as described in figure 6.

This walking appliance can be manufactured for about \$4.00 with aluminum as material.

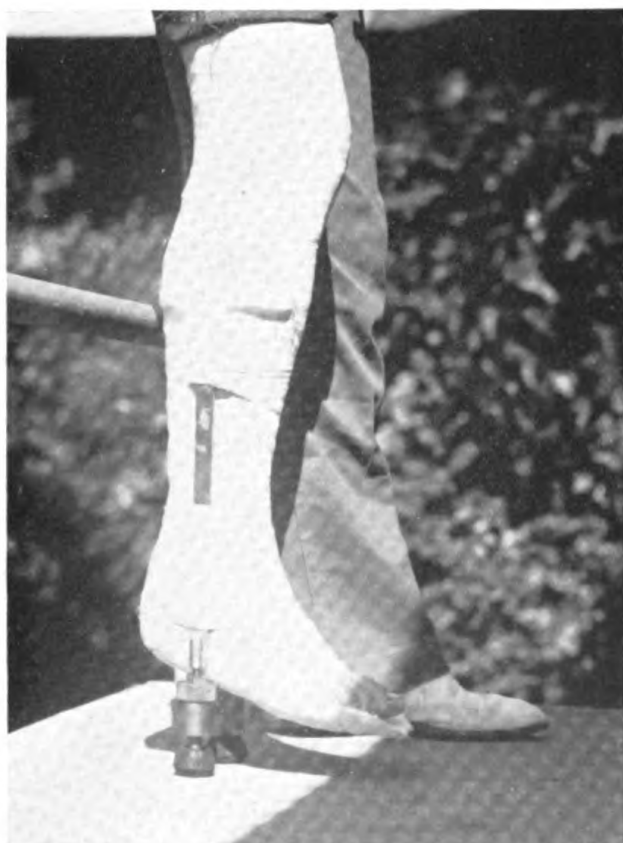


FIGURE 1.—WALKING APPLIANCE ASSEMBLED
SHOWING ITS APPLICATION TO A CAST.

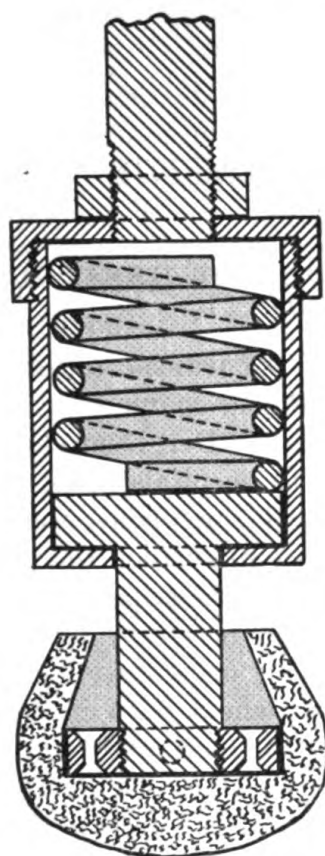


FIG. A1



FIG. 1

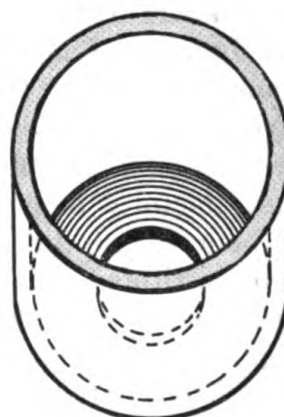


FIG. 2



FIG. 5

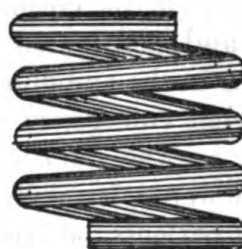


FIG. 3

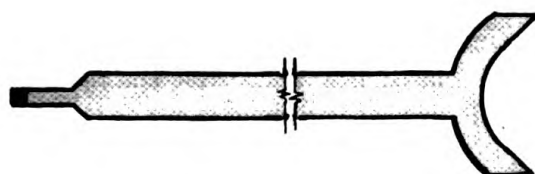


FIG. 6

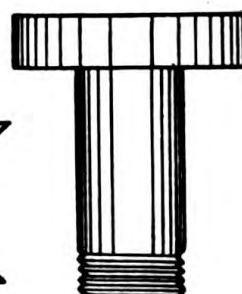


FIG. 4

NOTES AND COMMENTS

AIR TRANSPORTATION OF WOUNDED IN THE GERMAN CAMPAIGN IN POLAND

An interesting note on the method of transportation of wounded German soldiers during the recent Polish campaign is found in the German Journal of Military Medicine, "Der Deutsche Militararzt." Ambulance planes flew the patients to hospitals in Germany practically from the battlefield. On board the planes were emergency therapeutic agents as well as oxygen tanks which the medical officer could use, should it be necessary. Since the trips were usually short (2 to 3 hours), very little first aid was necessary after the original treatment instituted on the field. This organization made it possible for a soldier wounded on a battlefield deep in Poland in the morning, to be on an operating table in Germany by early afternoon. The method not only saved lives by lessening the delay between the time of injury and surgical interference, but decreased the duration of convalescence.

The Inspector of the Medical Service for the German Air Force established in the German Dental Corps a group of dental officers trained and designated for duty with the air ambulance service. They flew in the ambulance planes and were in charge of selecting the most urgent emergency face and jaw cases from those wounded and brought to the landing fields by automobile ambulances. The wounded were placed in planes which carried 8 to 12 beds and 8 to 12 seated patients, besides the medical officer. The rest of the wounded were left for selection by the medical officer in the next ambulance plane and this selection continued until all cases were evacuated.

Jaw fractures and face casualties were one type of injury that were given precedence for air transportation. The treatment of jaw fractures is considered of such importance that most hospitals have special departments for these casualties. Upon arrival, the wounded were rushed to these departments where they were cared for by dental surgeons. Early immobilization of jaw fractures, the use of an emergency splint within 24 to 48 hours, and a permanent splint 2 to 5 days after the injury resulted in optimum healing and recovery.

**MEETING OF THE AMERICAN COLLEGE OF PHYSICIANS
APRIL 21-25, 1941**

One of the features of the meeting of The American College of Physicians was a symposium on Military Medicine at which time the surgeons general of the Army, Navy, and Public Health Service appeared, together with several other speakers.

The Surgeon General of the Navy, Rear Admiral Ross T. McIntire, read a paper on "Problems of the Internist in the Navy." Commander C. S. Stephenson, Medical Corps, United States Navy, head of the Division of Preventive Medicine, Bureau of Medicine and Surgery, read a paper on "Special Medical Services in the Defense Program."

**MEDICAL OFFICERS ELECTED TO THE AMERICAN COLLEGE OF
PHYSICIANS FOR 1940-41**

The following medical officers of the Navy have been elected to Fellowship or Associateship of the American College of Physicians at the Boston meeting on April 20, 1941:

FELLOWSHIP

1. Commander Earl Richison, Medical Corps, United States Navy.
2. Lt. Comdr. Bartholomew W. Hogan, Medical Corps, United States Navy.
3. Lt. Comdr. Julian Love, Medical Corps, United States Navy.

ASSOCIATESHIP

1. Commander Frederick C. Greaves, Medical Corps, United States Navy.
2. Commander James F. Hays, Medical Corps, United States Navy.
3. Lt. Comdr. Hubert H. Carroll, Medical Corps, United States Navy.
4. Lt. Earl F. Evans, Medical Corps, United States Navy.
5. Lt. Frederick R. Lang, Medical Corps, United States Navy.
6. Lt. Robert A. Bell, Medical Corps, United States Navy.
7. Lt. Edgar M. Ricen, Medical Corps, United States Navy.

THE PREVENTION OF WAR WOUNDS BY ARMOR

Both in the World War and the present conflict, attempts to protect the soldier from either primary or secondary missiles has led to a return to the use of armor. In a sense the use of the tank is a reversion to the armored vehicles used in the Middle Ages in the assault of fortified places. Now bullet-proof shields for use by the individual soldier for certain assault duty has been recommended.

English and German military surgeons have observed that over one-third of the dead on battlefields had fatal chest wounds caused by splinters and secondary missiles of low penetrative power. Breast plates and armored jerkins capable of withstanding shrapnel, hand

grenade and bomb fragments, pistol bullets, and rifle bullets at extreme ranges have been not only advocated but devised and used.

Another device was a visor designed to furnish protection to the eyes. Cruse, in the *British Medical Journal* for May 18, 1940, says that during the first World War, 2,000 men were blinded in both eyes and 40,000 lost the sight of one eye in the British forces. He invented a visor of 22 gage duraluminum weighing 0.4 of a pound. It was designed to fit the inner curvature of the steel helmet to which it was attached by rivets and so arranged it could be lowered when occasion required. It was believed that such a device would have lessened the serious eye injuries by one-half.

The naval surgeon in the future may see the use of such individual armor with expeditionary forces ashore, and considering the high proportion of splinter wounds on cruisers, destroyers, and small craft, he may see it employed on board ship.

BOOK NOTICES

Publishers submitting books for review are requested to address them as follows:

The Editor:

UNITED STATES NAVAL MEDICAL BULLETIN,
Bureau of Medicine and Surgery, Navy Department.
Washington, D. C.

(For review)

THE ARMY MEDICAL SERVICES IN WAR, by *Lt. Col. T. B. Nicholls, M.B., Ch.B., Royal Army Medical Corps (Retired), medical officer, A. R. P., Derbyshire County, late deputy assistant director of hygiene and pathology, Northern Ireland District, assistant director of hygiene, southern command, India, assistant director of hygiene and pathology, western command; with chapters by Air-Commodore A. S. Glynn, M.B., Ch.B., K.H.S., R.A.F., P.M.O., Fighter Command R. A. F.; Col. A. R. Laurie, M.B., Ch.B., D.M.R.E. (T.A.), A.D.M.S., Second Antiaircraft Division, and Col. F. G. Lescher, M.C., M.A., M.D., M.R.C.P. (T.A.), group officer, E. M. S.* Second edition, pp. 488, Williams & Wilkins Co., Baltimore, Md., publishers, 1940. Price \$5.00.

This book was prepared for the British army medical officer as a "study for understanding the problems of the medical services in the field, and for preparing for their promotion examinations." This second edition was published three years after the first, because the latter was sold out and because the present war in Europe recreated new problems in medical services.

The subject matter is well chosen and arranged. The author's style is concise and never verbose. Parts I, II, and III covering "General Organization and Administration," "Constitution and Organization of Medical Units," and "Strategy and Tactics," respectively, contain invaluable information for the Navy medical officer serving with our Fleet Marine Force. Part IV describes the organization and function of the present Emergency Medical Service in the British Isles and is of particular interest to medical administrators of hospitals. The last part contains illustrative medical military problems. These, no doubt, would be of most value to medical officers engaged in the teaching of medical tactics.

STATE BOARD QUESTIONS AND ANSWERS FOR NURSES, Essay and Objective Types.

Compiled from actual examination questions given throughout the country by State examining boards. Nineteenth edition, revised. Edited by 11 authorities in nursing education. J. B. Lippincott Co., Philadelphia, Pa., publishers, 1941. Price \$3.50.

The new 1941 revised edition presents every subject in the basic course for nurses. It is divided into four parts: Biologic and Physical Science; Medical Science; Nursing and Allied Arts; History and Ethics of Nursing with essay and objective type questions and answers, including true and false, completion, matching terms, selection—single and multiple type, etc.

This textbook should prove invaluable to the student as an aid in the systematic study for examinations, and to the graduate it is an ideal way in which to review the nursing procedures and acquaint herself with the latest developments in the nursing world. The instructor will likewise find it a ready guide in the preparation of written and oral tests.

This book should, by all means, be included in all hospital libraries.

THE 1940 YEAR BOOKS, published by The Year Book Publishers, Inc., 304 South Dearborn Street, Chicago, Ill.

These year books are so well known to medical men that little more is required than to bring the books to their notice by publishing the list of this year's volumes. Each contains the latest information on new developments in each field by recognized specialists. The books comprising the 1940 series are listed below:

Subject	Authors	Price
Obstetrics and Gynecology-----	{ Joseph B. DeLee, M. D.-----	\$2. 50
General Therapeutics-----	{ J. P. Greenhill, M. D.-----	
	Oscar W. Bethea, M. D.-----	2. 50
Neurology, Psychiatry, and Endocrinology.	{ Hans H. Reese, M. D.-----	3. 00
	{ Nolan D. C. Lewis, M. D.-----	
Pathology and Immunology-----	{ Elmer L. Sevringhaus, M. D.-----	3. 00
Industrial and Orthopedic Surgery.	{ Howard T. Karsner, M. D.-----	
Public Health-----	{ Sanford B. Hooker, M. D.-----	3. 00
Radiology-----	{ Charles F. Painter, M. D.-----	
Urology-----	{ J. C. Geiger, M. D.-----	5. 00
General Surgery-----	{ Charles A. Waters, M. D.-----	
Eye, Ear, Nose, and Throat-----	{ Ira I. Kaplan, M. D.-----	3. 00
Physical Therapy-----	{ Oswald S. Lowsley, M. D.-----	
Dermatology and Syphilology----	{ Evarts A. Graham, M. D.-----	3. 00
	{ Louis Bothman, M. D.-----	
	{ Samuel J. Crowe, M. D.-----	2. 50
	{ Richard Kovacs, M. D.-----	
	{ Marion B. Sulzberger, M. D.-----	3. 00
	{ Fred Wise, M. D.-----	
	{ George F. Dick, M. D.-----	3. 00
	{ J. Burns Amberson, Jr., M. D.-----	
General Medicine-----	{ George R. Minot, M. D.-----	3. 00
	{ William B. Castle, M. D.-----	
	{ William D. Stroud, M. D.-----	3. 00
	{ George B. Eusterman, M. D.-----	

Subject	Authors	Price
Pediatrics.....	{ Isaac A. Abt, M. D.....	\$2. 50
	{ Arthur F. Abt, M. D.....	
	{ Charles G. Darlington, M. D.....	
Dentistry.....	{ George W. Wilson, D. D. S.....	3. 00
	{ Howard C. Miller, D. D. S.....	
	{ Walter H. Wright, D. D. S.....	
	{ George R. Moore, D. D. S.....	

HISTORY OF PHARMACY, A Guide and a Survey by *Edward Kremers, Ph.G., Ph.M., Ph.D., Sc.D.,* former director, course in pharmacy and professor of pharmaceutical chemistry, University of Wisconsin; author, editor and historian; and *George Urdang, Ph.G., D.Sc.Nat.,* honorary member of the American Pharmaceutical Association; former editor of the *Pharmazeutische Zeitung*; former director of the Society for the History of Pharmacy, Berlin; author and historian. Pp. 468, 30 illustrations. J. B. Lippincott Co., Philadelphia, Pa., publishers, 1940. Price \$4.50.

It is a noteworthy achievement to write a history of pharmacy in a single volume of 466 pages. This work presents a complete picture of the development of pharmacy from the dawn of civilization to the twentieth century. There has been included the background of cultural and political history which is so necessary to a proper interpretation of trends and movements in pharmacy.

The material available for a book of this kind was extensive, but at the same time incomplete and scattered. The task to select and coordinate this mass of material in order to meet the needs of pharmacy students and lecturers was arduous. But the authors have succeeded in preparing a book admirably suited for use both as a textbook and for general reading.

The book is divided into 4 parts. Part 1—Early background in the old world which includes the ancient civilization in Babylon-Assyria, Egypt, Greece, and Rome. Part 2—The rise of professional pharmacy in Europe embracing international trends, development of pharmacy in Italy, France, Germany, and England. Part 3—Pharmacy in the United States from early Colonial period, through the growth of associations, the rise of legislative regulation, the development of education, the establishment of a pharmaceutical literature to the present day economic structure of pharmacy. Part 4—Discoveries, inventions, and other contributions to society by pharmacists.

This book has an extensive bibliography (over 1,000 references) and a compact and well organized glossary. These features will greatly assist students in pharmaceutical research. The illustrations are few but pertinent and interesting. The type is large and clear. The index is complete.

ESSENTIALS OF THE DIAGNOSTIC EXAMINATION, by *John B. Youmans, B.A., M.S., M.D.*, associate professor of medicine and director of postgraduate instruction, *Vanderbilt University Medical School*. 417 pages. The Commonwealth Fund, New York, N. Y. 1940. Price \$3.00.

This handbook consists of three parts. Part 1, the history and physical examination; Part 2, laboratory tests; Part 3, miscellaneous tests, including the essential equipment for an office laboratory with the chemicals, reagents and stains, together with a list of books suggested for additional reading for those who may wish more detailed information concerning the various methods of examinations.

As stated by the author in the preface: "This book has been written on the basis of the experience the author gained while teaching physical diagnosis and clinical laboratory methods to practitioners during the past 10 years, and is in the main an expansion of the outlines used in those courses. Its purpose is to assemble in a single volume the minimum of diagnostic methods and procedures needed in the general practice of medicine and available to all physicians."

The author has succeeded admirably in carrying out this purpose. The sections on cardiac and neurological examination are particularly good as is the laboratory section, which gives the physiologic background of the tests, indications for their use, and their diagnostic significance.

"Ovale tertian" malaria is not mentioned. It is considered that the importance of using the bell of the stethoscope in listening for the low-pitched rumbling mid or late diastolic murmur of mitral stenosis as contrasted with the diaphragm of the stethoscope in detecting the early, soft blowing, diastolic murmur of aortic insufficiency should be emphasized as well as the value of change in position, full expiration and full inspiration. The value of having the patient lean well forward in full expiration in bringing out the systolic thrill of aortic stenosis also seems underemphasized.

Regardless of these minor criticisms, this handbook is considered to be a very valuable aid to the physician in his daily practice and an especially valuable handbook for internes.

DISEASES OF THE DIGESTIVE SYSTEM, A Text-book for Students and Practitioners, by *Eugene Rosenthal, M.D.*, lecturer in the medical faculty, *Royal Peter Pazmany University, Budapest, Hungary*, with a preface by *R. J. V. Palvertaft, M.D., F.R.C.P.*, reader in pathology, *University of London*; director of the *John Burford Carlill Laboratories and curator of museum, Westminster Hospital School of Medicine*. With 234 illustrations, including 104 in color, and 16 tables. 394 tables. C. V. Mosby Co., publishers, 1940. Price \$8.50.

The author, a clinical teacher of distinction in Budapest, presents the essentials of the pathology, diagnosis and therapy of the digestive organs with a liberal use of diagrams. The illustrations and diagrams number 234. They deal with the common gastro-intestinal

disorders of general practice and for a student with a strong visual memory, will leave a clear picture which he can apply to his patient. Diseases of the stomach and of the liver and biliary tract are especially well described and in each section the careful attention given to the physical examination is to be commended. One misses a discussion of the Miller-Abbott tube under intestinal obstruction, as well as a bibliography. The binding, quality of paper, and type are excellent.

BACTERIOLOGY OF PUBLIC HEALTH by *George M. Cameron, Ph.D., associate professor of bacteriology, University of Tennessee.* 451 pages, illustrated. C. V. Mosby Co., St. Louis, publishers, 1940. Price \$3.50.

Elementary work dealing with pathogenic bacteriology as related to public health. This book may be recommended to serve as an introductory text to a college course in bacteriology and as of interest to the layman.

No attempt was made to describe laboratory technic but it briefly gives descriptions of disease processes which are of value in understanding infecting organisms.

The book is well bound and typed and the author's style leads to easy reading.

PHARMACOLOGY AND THERAPEUTICS, by *Arthur R. Cushny, M.A., M.D., L.L.D., F.R.S., late professor materia medica and pharmacology in the University of Edinburgh.* Thoroughly revised by *C. W. Edmunds, A.B., M.D., professor of materia medica and therapeutics in the University of Michigan, Ann Arbor, Michigan,* and *J. A. Gunn, M.D., D.Sc., F.R.C.P., professor of pharmacology in the University of Oxford, Oxford, England.* Twelfth edition. 852 pages, illustrated with 66 engravings. Lea & Febiger, Philadelphia, publishers, 1940. Price \$6.50.

The first edition of this book appeared in 1899; the twelfth edition appeared in 1940. During the intervening years it has continued to serve as a critical, scientific, general textbook on pharmacology. The preparation of the ninth, tenth, eleventh, and present editions was entrusted to Doctors Edmunds and Gunn who have contributed much of their own knowledge and experience to make this book outstanding among textbooks on this important subject.

The twelfth edition includes the changes required by the first and second supplements of the eleventh revision of the United States Pharmacopoeia and the appendix to the British Pharmacopoeia of 1936.

Many new pharmacological substances, especially new synthetic compounds leave research laboratories every year. Some of them show little or no promise of therapeutic use. The authors of the twelfth edition of this work, with a fine sense of discernment, have emphasized only those medicinal agents which have been found to be of therapeutic importance. Consequently this book is well suited as a textbook for medical students and practitioners.

Besides the introductory section, this book is divided into six parts. The first part is devoted to a discussion of the action of inorganic substances. Part 2 deals with substances characterized chiefly by their local action. Part 3 includes substances characterized chiefly by their action after absorption. Part 4 emphasizes anthelmintics. Part 5 covers the important antiseptics and disinfectants. Part 6, the concluding section, discusses the therapeutic effects of vaccines, toxins, and antitoxins. The classification of drugs according to their therapeutic uses is well arranged and should prove valuable to physicians in writing prescriptions.

The book is well printed, well indexed and the bibliography is sufficiently complete to meet the needs of medical students and physicians.

FOREIGN BODIES LEFT IN THE ABDOMEN, by *Harry Sturgeon Crossen, M.D., School of Medicine, Washington University, St. Louis, Mo., and David Frederic Crossen, LL.B., School of Law, Washington University, St. Louis, Mo.* Pp. 762, with 212 illustrations including 4 color plates. C. V. Mosby Co., St. Louis, publishers, 1940. Price \$10.00.

Any surgeon who has lived through the suspense accompanying the search for a lost sponge after the abdomen has been closed will appreciate this work. It is an unusual book in many respects and having been written jointly by a surgeon and a lawyer, the legal as well as the medical aspects of the subject are covered.

The authors are of the opinion that this type of accident occurs much more frequently than is generally supposed, as many cases do not get into the literature. Of the several hundred cases reported, more than 300 were of sponges left in the abdomen at the time of laparotomy. The seriousness of this accident is revealed by their collected statistics which show that approximately one-fourth of the cases terminated fatally.

The pathology associated with foreign bodies is discussed at length and the symptoms and signs are presented for the different time-period groups. Each type of accident has been grouped so that all of the existing information on the subject is made accessible to the reader. Treatment is outlined and much space is given over to prevention, the various methods of sponge control being described in detail.

This book represents an immense amount of work in searching, analyzing, and arranging the material culled from the known literature. The printing and binding are first-rate and the bibliography is complete.

Written on a subject that most surgeons prefer not to think about, this book once picked up is hard to put down. It is both interesting and instructive and deserves a place in every hospital and surgeon's

library as a grim reminder that such accidents happen, and when they do, they frequently rob the patient of his inherent right to live.

METHODS FOR DIAGNOSTIC BACTERIOLOGY, A Complete Guide for the Isolation and Identification of Pathogenic Bacteria for Medical Bacteriology Laboratories, by *Isabelle G. Schaub, A.B., assistant in bacteriology, department of pathology and bacteriology, The Johns Hopkins University School of Medicine; and M. Kathleen Foley, A.B., bacteriologist in charge of the Diagnostic Bacteriological Laboratory of the Medical Clinic, The Johns Hopkins Hospital, Baltimore.* Pp. 313. C. V. Mosby Co., St. Louis, Mo., publishers, 1940. Price \$3.00.

This practical and valuable book represents the experience of the authors in teaching concise, simplified technics of diagnostic bacteriology to technicians who have mastered a few fundamentals of bacteriological technics. Explicit directions are given for the handling of clinical and autopsy material and day-by-day directions for identification of organisms which are commonly encountered in medical bacteriology. Methods for the serological study of organisms and of patients' sera, also preparation of culture media and reagents, are given.

APPLIED PHARMACOLOGY, by *Hugh Alister McGuigan, Ph.D., M.D., F.A.C.P., professor of pharmacology and therapeutics, University of Illinois, College of Medicine.* 914 pages, illustrated. C. V. Mosby Co., publishers, 1940. Price \$9.00.

This book of 914 pages and 41 selected illustrations is a worthy contribution to the science of pharmacology. It is an excellent textbook for medical students and admirable as a reference for medical officers. The subject matter is well arranged in 84 topical subdivisions with a comprehensive bibliography following the more important sections. This edition conforms to the eleventh revision of the United States Pharmacopeia and the sixth revision of the National Formulary. The British Pharmacopeia and Codex are quoted.

The many subjects—physiology, histology, therapeutics, and biochemistry—have been well covered in this book and there are many outstanding sections. However, the discussion of "Anesthetics and Narcotics," including a brief historical outline of the various theories and development of anesthesia, merit special comment. The material in the sections on "The Chemotherapy of Syphilis" and "Vitamines" is well organized and interestingly developed. The chapter on "Prescription Writing" is important to the medical student and the pages devoted to the "Solubilities of the Common Substances" will no doubt prove to be of inestimable value to the physician in prescription writing. The index has been carefully prepared.

AN INTRODUCTION TO BIOCHEMISTRY, by *William Robert Fearon, M.A., S.D., M.B., F.I.C., fellow of Trinity College, Dublin; member of the Royal Irish Academy.* Pp. 475, second edition, C. V. Mosby Co., St. Louis, Mo., publishers, 1940. Price \$3.75.

This edition is a small, concise, well-written, up-to-date book which stresses the chemical rather than the physiological approach to the fundamentals of biochemistry. Recent advances in this field have necessitated an extensive revision of the subject matter and the addition of new chapters on solutions, colloidal systems, pigments, steroids, tissue respiration and internal environment. This edition has undergone an excellent and thorough revision which makes it up-to-date in every respect.

The book is intended primarily for the student but anyone desiring a review of the fundamentals of biochemistry should find it especially valuable.

THE FUNDAMENTALS OF NUTRITION, by *Estelle E. Hawley, Ph.D., and Esther E. Maurer-Mast, M.D., University of Rochester, School of Medicine and Dentistry, Rochester, N. Y., including table of 100-calorie portions by Estelle E. Hawley, Esther E. Maurer, and Herbert F. Van Epps, Department of Vital Economics, University of Rochester, and discussions of the dietary management in specific conditions by collaborators associated or formerly associated with The University of Rochester, School of Medicine and Dentistry, with a foreword by John R. Murlin, Ph.D., Sc.D., professor of physiology and director of the Department of Vital Economics, University of Rochester.* 477 pages, illustrated. Charles C. Thomas, Baltimore, Md., publishers, 1940. Price \$5.00.

The authors present first the fundamentals of energy metabolism and of the normal diet in the light of recent knowledge especially relating to vitamins and minerals. Diet planning centers around a table of 100-calorie portions of food, since the common servings of so many foods yield about 100 calories. This 100-calorie unit does not seem to have any real practical advantages. Many tables, both in the text and in the appendix, give the composition of foods with respect not only to carbohydrates, protein, and fat but also to minerals, vitamins, and other essentials.

Tabulation of the normal constituents of blood, urine, and feces and laboratory methods for determining vitamin values are also included.

A group of 19 contributors have provided discussions of the dietary managements of such specific conditions as obesity, diabetes, hyperinsulinism, gout, gastro-intestinal diseases, anemia, toxemias of pregnancy, food allergy, surgery, urolithiasis, and vitamin deficiencies. Extensive bibliographies are given with some regrettable typographical errors. The volume is well printed and bound.

THE ESSENTIALS OF PHYSIOLOGY AND PHARMACODYNAMICS, by *George Bachmann, M.S., M.D., professor of physiology in the School of Medicine of Emory University; sometime demonstrator of physiology in the Jefferson Medical College of Philadelphia, and A. Richard Bliss, Jr., Ph.D., M.D., LL.D., professor of pharmacology and dean of pharmacy in Howard College of Birmingham, Alabama, formerly chief of division of pharmacology in College of Medicine of the University of Tennessee and in School of Medicine of Emory University; director of the physiology and pharmacodynamics studies of the Commonwealth Fund Study of Pharmacy from the Functional Standpoint; member of committee of revision of the U. S. Pharmacopoeia.* Third edition, revised and reset, pp. 508, The Blakiston Co., Philadelphia, Pa., publishers, 1940. Price \$4.50.

There has been a definite need for a textbook on physiology and pharmacodynamics which is especially suited for students of pharmacy. The usual textbooks on these subjects prepared for medical students are not entirely satisfactory since they are too advanced. The smaller texts prepared for students in nursing, general science, and physical education, it is believed, are too elementary and for that reason not very well adapted to meet the needs of pharmacy students. The authors have succeeded in preparing an excellent textbook on physiology and pharmacodynamics which meet the requirements of the pharmaceutical syllabus.

The third edition has been almost completely rewritten due to recent advances in physiology and pharmacodynamics and includes the changes necessitated by the latest revision of the United States Pharmacopoeia, National Formulary, New and Nonofficial Remedies, and the British Pharmacopoeia. In the revision the authors have emphasized those topics in which there have been important changes, as—the chemistry of muscle contraction; the nature, velocity, and frequency of discharge of the nerve impulse; the pharmacodynamics of the cell; allergic protein extracts; serums and vaccines; endocrine compounds; etc.

Certain portions of anatomy and the histology of the epithelial and connective tissues necessary for the teaching of physiology have been included to give the pharmacy student sufficient basis for a clear understanding of physiology. The 196 illustrations will materially assist the student in the study of this important subject.

A TEXTBOOK OF HISTOLOGY, by *Harvey Ernest Jordan, A.M., Ph.D., professor of anatomy and director of the Anatomical Laboratories, University of Virginia.* Eighth edition, pp. 690. D. Appleton-Century Co., New York, publishers, 1940. Price, \$7.00.

In the eighth edition of this excellent histology the chapters on Blood, Muscle, the Endocrine Organs, the Female Reproductive System and the Blood Vascular System have been thoroughly revised.

References are recorded as footnotes and reassembled in an extensive bibliographical list at the end of the book. The text is clear and concise and well illustrated.

THE ESSENTIALS OF APPLIED MEDICAL LABORATORY TECHNIC, Details of How to Build and Conduct an Office or Small Hospital Laboratory at Small Cost, by *J. F. Feder, M.D., director of laboratories and allergic service, Anderson County Hospital, Anderson, S. C.* 241 pages, profusely illustrated, two plates in colors. Charlotte Medical Press, Charlotte, N. C., 1940. Price, \$5.00.

This book gives the details of how to build and conduct an office or small hospital laboratory at small cost. It has been written with the end in view to furnish in plain terms to the office nurse technician and the smaller laboratories some fundamental instruction in the essential features of laboratory technic. It will be found very helpful in this field. There are many helpful illustrations and a chapter on Blood and Plasma Transfusion by Dr. John Elliott.

CERAMICS IN DENTISTRY, by *Milton Cohen, D.D.S., F.I.C.D., formerly professor of crown and bridgework and clinical professor of dental ceramics and ceramic technology, New York University, College of Dentistry, New York City.* Imperial octavo, 335 pages, illustrated with 707 engravings on 696 figures, 26 in colors. Lea & Febiger, Philadelphia, Pa., publishers, 1940. Price, \$10.00.

This is an excellent book, comprised of 9 chapters containing 335 pages. It is based upon the accumulated practical experience of the author over a period of 25 years.

The subject matter is presented in a clear and precise manner and illustrated with 696 photographs and diagrammatic drawings. These illustrations are beautifully done and are well selected and arranged.

This text is recommended to both the general practitioner and one specializing in dental ceramics.

THE SCIENCE OF DENTAL MATERIALS, by *Eugene W. Skinner, Ph.D., associate professor of physics, Northwestern University Dental School.* Second edition, revised, 421 pages, illus. W. B. Saunders Co., Philadelphia, publishers, 1940. Price \$4.75.

To know the materials with which one works is essential to an intelligent understanding of the finished structure, whether it be a building or a filling.

Dr. Skinner's book supplies this information in the form of a valuable compilation of the results of research with dental materials. The reader is given data with which he may better evaluate these materials on a scientific basis in regard to their sources, properties, composition, and manipulation.

This work is unique in character as it represents the only one of its kind which is devoted to comprehensively covering the field of the most frequently used dental materials. In addition to serving as a splendid textbook as a part of the dental curriculum, it should

be much appreciated by the progressive practitioner who is zealous of keeping himself well informed in order to render the best service to his patients.

This volume is well written, broad in its scope, and adequately supplied with photographs, tables and graphs explanatory of the text. Of the 24 chapters, those dealing with the acrylic resins and the steels are especially timely, inasmuch as they present the more recent findings in regard to these much-discussed newer dental materials.

The appendix gives the American Dental Association specifications for certain impression materials, investments, waxes, amalgams, golds, and cements.

A wealth of information is contained between the covers of this book, which should provide a ready means of study and reference.

THE TREATMENT OF DIABETES MELLITUS, by *Elliott P. Joslin, A.M., M.D., Sc.D.*, medical director, *George F. Baker Clinic, New England Deaconess Hospital*; clinical professor of medicine emeritus, *Harvard Medical School*; consulting physician, *Boston City Hospital*. *Howard F. Root, M.D.*, physician, *New England Deaconess Hospital*; consultant in medicine, *Eastern Maine General Hospital*; *Massachusetts State Infirmary, Tewksbury, Middlesex County Tuberculosis Sanitarium*; instructor in medicine, *Harvard Medical School*. *Priscilla White, M.D.*, physician, *New England Deaconess Hospital*; instructor in pediatrics, *Tufts College Medical School*; and *Alexander Marble, A.M., M.D.*, physician, *New England Deaconess Hospital*; instructor in medicine, *Harvard Medical School*. Seventh edition, 783 pages, illustrated. Lea & Febiger, Philadelphia, Pa., publishers, 1940. Price \$7.50.

The seventh edition of this work on the treatment of diabetes makes it again the outstanding work on the subject.

With Dr. Joslin, this edition is under authorship of Drs. Root, White, and Marble. These associates of the senior author have for years been laboring with him in the clinic and laboratory in the study and treatment of diabetes. Through these years our knowledge of the subject has steadily expanded and at no time has it made more rapid advances than in the period covered by this edition. These experts in the several phases of this disease now bring their special knowledge and experience directly to us in the revision of the various chapters.

All the recent advances have been carefully reviewed and evaluated. With these discussions a most complete bibliography of recent literature has been given. This will prove valuable for reading in any line.

The new and probably epoch making work of Houssay and Evans with the discoveries of Young on the role of the pituitary gland in the causation of diabetes in experimental animals is given. The

application of this new concept to the etiology, prevention, and treatment of human diabetes, especially the juvenile type, is indicated. New chapters on allergy and on hyperinsulinism have been introduced. Insulin reactions have again received the emphasis they deserve, for, as Joslin says, though coma may end a diabetic life, insulin reactions will wreck it. The value and practical points in the use of the various forms of insulin available are thoroughly discussed. Changes in the management of the diabetic under protamine insulin make us realize why Joslin has called the period since the advent of this insulin the "Hagedorn Era."

In studying this book one comes to realize that the diabetic is an individual in whom all the phases of medicine, from pediatrics to geriatrics, from metabolism to endocrinology, present different problems than in the nondiabetic. A thorough knowledge of diabetes, then, includes a fine background in all branches of medicine.

NEWER NUTRITION IN PEDIATRIC PRACTICE, by *I. Newton Kugelmass, B.S., M.A., M.D., Ph.D., Sc.D.*, attending pediatrician, Broad Street Hospital, New York, and Heckscher Institute, New York; consultant pediatrician Lynn Memorial Hospital, Monmouth Memorial Hospital and Muhlenberg Hospital, N. J.; formerly visiting pediatrician French Hospital and New York City Childrens Hospital; director pediatric research, Fifth Avenue Hospital; pediatric research associate, Yale University. Pp. 1155 pages. J. B. Lippincott Co., Philadelphia, Pa., publishers, 1940. Price \$10.00.

This book is an invaluable aid to the practitioner of pediatrics. The logical and comprehensive presentation of material by division into the three units—(1) nutritional physiology, (2) nutrition in health, and (3) nutrition in disease, renders it easy for the busy and active physician to review the particular problem in which he is interested with the least utilization of time and effort. The analysis of material presented is positive rather than indefinite or negative, and points out the practical application for effective nutritional therapy, both qualitatively and quantitatively; as well as giving established principles and procedures for the prevention of disease and the advancement of health. The latter phase is of paramount importance in the pediatric field because of the concentration of interest along preventive lines.

Further, the presentation of nutritional information in specific diseases as to the clinical concept, etiologic factors, metabolic mechanism, pathologic anatomy, clinical features, differential diagnosis, and therapeutic procedures gives a clear-cut and concise picture of the disease entity which may be readily visualized—a feature of paramount importance in the day-by-day practice of pediatrics. The tables presented and the illustrations given are excellent and informative.

MANAGEMENT OF THE CARDIAC PATIENT, by William G. Leaman, Jr., M.D., F.A.C.P., *assistant professor of medicine in charge of the department of cardiology, Women's Medical College of Pennsylvania, Philadelphia; cardiologist, Woman's College, Memorial, Northeastern Hospitals and Philadelphia Hospital for Contagious Diseases; consulting cardiologist, St. Luke's and Children's Hospital, Philadelphia; assistant visiting physician, Philadelphia General Hospital; chairman, committee on diseases of the heart and circulation, Philadelphia County Medical Association. Fellow, College of Physicians of Philadelphia.* Pp. 705, 255 original illustrations. J. B. Lippincott Co., Philadelphia, publishers, 1940. Price \$6.50.

As the author states in his preface, this book was written for the general practitioner. Facts most essential in the management of cardiac patients are assembled in 657 pages of text. There are numerous diagrams, drawings, photographs of autopsy material, reduced copies of x-ray films and reproductions of electrocardiograms. Roentgen methods in diagnosis are briefly reviewed. For those unfamiliar with electrocardiography a final chapter gives the basic information.

The diagnosis and treatment of a large number of cases are outlined to offer the reader an example of every cardiovascular problem which he may expect to meet. A discussion following each case history emphasizes the significant features.

A practitioner after complete reading of this book may be assured that he has been informed of all the facts essential in the management of cardiac patients. The illustrative case histories with discussions may later serve as quick references for comparison with cases of his own.

A TREATISE ON MEDICOLEGAL OPHTHALMOLOGY, by Albert C. Snell, M.D., *lecturer in ophthalmology, School of Medicine and Dentistry, University of Rochester; consultant in ophthalmology, Strong Memorial Hospital, and Rochester General Hospital; ophthalmologist, Park Avenue Hospital, Rochester, New York; member of the American Ophthalmological Society; Fellow of the American College of Surgeons, the American Medical Association, and the American Academy of Ophthalmology and Otolaryngology.* Pp. 312, illustrated. C. V. Mosby Co., St. Louis, Mo, publishers, 1940. Price \$6.00.

In this treatise the author attempts to present all phases of medico-legal ophthalmology. In the first part of the book, he reviews some of the problems of a medical witness on the stand in court and offers sound advice on procedure. The physician-patient relationship is also discussed at some length and the rights of both are delineated. Malpractice and its avoidance are dealt with to some extent.

After giving a review of the historical development of compensation laws in this country and abroad, the author develops his chief theme, that is, the proper evaluation of loss of function of the eyes and a proper reward of compensation for such loss. The flaws in existing statutes of various states are pointed out and an ideal form of statute is outlined. The contention is made that an estimate

of the percentage loss of function should take into account: central visual acuity for distance and near, peripheral vision, and muscle function of the eye. All estimates of loss of visual acuity should be based on the best possible vision obtainable with corrective lenses.

Methods of examination of compensation cases are discussed, and the detection of malingerers is well explained. Standard methods are suggested for the evaluation of visual disabilities, taking all factors into consideration and using as a basic principle the functional efficiency of the eye.

In the appendix many excerpts from court decisions on compensable ocular disabilities are given.

The text is well written with few highly technical discussions.

The binding is of good quality; the paper is well chosen for a book on ophthalmology with glare largely eliminated.

THE DIVISION OF PREVENTIVE MEDICINE

Captain C. S. Stephenson, Medical Corps, United States Navy, in charge

AN ANALYSIS OF TOE INJURIES IN RELATION TO ACCIDENT PREVENTION IN A NAVY YARD¹

By Lieutenant Hyman Lieber, Medical Corps, United States Naval Reserve

INTRODUCTION

This report presents an analysis of all cases of severe toe injury during the period of 1 year ending November 22, 1940. The findings are of interest for the following reasons:

- (1) As demonstrating the financial and production time loss which could have been largely prevented by means of an available safeguard.
- (2) As indicating a marked difference in hazard among the various trades in the yard.
- (3) In serving as a basis of comparison for future period of observation.

OBJECTIVES OF THE STUDY

The cases are analyzed under the following headings:

- (a) Frequency of fractured toes.
- (b) Frequency of contused toe injuries involving lost time.
- (c) Time loss from fractured toes.
- (d) Distribution of fracture cases by occupations.
- (e) Distribution of fracture cases by shops.
- (f) Comparative hazard of broken toes in various trades.
- (g) Seasonal distribution of toe fractures.
- (h) Site of fracture as determined by X-ray examination.
- (i) Financial aspects of toe injuries.
- (j) Prevention of toe injuries.
- (k) Recommendations.

FREQUENCY OF BROKEN TOES

Ninety-nine cases of fractured toes were seen at the navy yard dispensary in 1 year. Five men were not regular yard employees. Four were W. P. A. workers, and one was employed by a private contractor on a yard project. Only the 94 cases involving regular yard employees were analyzed for time loss, etc. These men represent 0.83 percent of the average number of industrial employees during the period of this study.

¹ From November 22, 1939, to November 21, 1940.

One out of every 115 workers at this navy yard broke a toe during the year.

FREQUENCY OF CONTUSED TOE INJURIES INVOLVING LOST TIME

Nineteen instances of contusion of the toes were so severe as to require loss of time. These cases caused a total loss of 95 working days during the year. When these cases are added to those cited under the previous heading, we find 1 out of every 96 industrial workers suffered a lost-time injury during the year.

The monthly accident reports of the yard show that there were 930 lost-time accidents from all causes during the year. The 93 toe injuries that required loss of time accounted for exactly 10 percent of all the tabulatable accidents for the year.

TIME-LOSS FROM FRACTURED TOES

Seventy-four of the men with fractured toes lost a total of 1,056 working days. The average number of days lost per man was 14.3. The longest disability was 42 days. This occurred in two cases. Twenty men continued working despite their broken toes, most of them at light duties. No attempt has been made to assess the loss of efficiency in this group. The total time lost for the combined group of fractures and contusions was 1,153 days.

Of the total number of working days lost at this navy yard during the 12 months covered in this report, 22,500 days were the statistical charges for three deaths and an amputation through the thigh. If these are deducted from the total of 32,125 days, we find that 9,625 working days were lost because of common injuries. The 1,153 days loss incident to toe injuries represent 12 percent of this entire amount.

TABLE 1.—*Distribution of fracture cases by occupations*

Rigger	10	Holder-on	2
Rigger's helper	4	Joiner	2
Shipfitter	2	General helper	2
Shipfitter helper	7	Boatbuilder	2
Machinist	6	Blacksmith helper	2
Machinist helper	3	Boilermaker	1
Laborer	7	Boilermaker helper	1
Driller	7	Firewatch	1
Shipwright	7	Flange turner	1
Sheet-metal worker	6	Brakeman	1
Pipefitter	2	Painter	1
Pipefitter helper	3	Millman	1
Welder	4	Packer	1
Plumber	2	Coppersmith helper	1
Plumber helper	1	Electrician and molder helper	1
Chipper and caulker	3		

It will be noted that 31 occupations were involved. The highest incidence was among riggers, i. e., 10.6 percent. No significant prevalence occurred in the remaining occupations.

TABLE 2.—*Distribution of fracture cases by shops*

	<i>Shop No. Cases</i>			<i>Shop No. Cases</i>	
Laborer	01	5	Machinist.....	38	2
Transportation	02	1	Boilermaker	41	1
Shipfitter	11	3	Electrician	51	1
Do	12	8	Coppersmith	53	1
Do	13	1	Pipefitting	56	8
Do	15	5	Shipwright	61	10
Do	16	6	Joiner	62	1
Sheet Metal	17	6	Paint	71	1
Shipsmith	23	2	Rigger	72	16
Welding.....	26	2	Building trades.....	79	3
Do	27	2	Foundry	82	1
Machinist	31	6			—
Do	32	1			94
Do	36	1			

It will be noted that there is a marked variation in incidence among the several shops in one trade. Six of the 10 cases among machinists occurred in shop 31; the remaining 4 cases were found in 3 machine shops.

TABLE 3.—*Comparative hazard of broken toes in various trades*

Trade	Average number employed	Fractures	Incidence (percent)
Shipfitters ¹	2502	9	0.36
Machinists ¹	1934	9	.47
Welders.....	677	4	.59
Plumbers ¹	661	3	.45
Laborers.....	638	7	1.10
Sheet-metal workers.....	551	6	1.09
Riggers.....	457	14	3.06
Joiners.....	557	2	.36

¹ Includes helpers.

Marked variations in incidence are apparent from table 3. More than 3 percent of the riggers employed in this yard were disabled by fractured toes in 1 year. The shipfitters have only one-tenth as many broken toes as riggers. Other occupations fall between these extremes, and the figures of table 3 may be compared with the average incidence of 0.83 percent for all the industrial workers of the yard.

SEASONAL DISTRIBUTION OF TOE FRACTURES

Analysis of the injuries by months shows a peculiar increase in frequency of toe injuries during the summer. In the 5 months from April to August, inclusive, there were 54 cases, but in the remaining 7 months there were only 40 cases. Since these figures run counter to the curve of employment, they may be considered significant. One reason for this seasonal increase might be the tendency of the workers to wear lighter footgear during the summer months.

SITE OF FRACTURE AS DETERMINED BY X-RAY EXAMINATION

Complete x-ray studies were made in 91 cases. In several cases more than one toe bone was fractured.

TABLE 4.—*Classification according to anatomical distribution*

	Tip	Center	Base
Great toe.....	67	10	8
Second toe.....	9	1	0
Third toe.....	5	0	2
Fourth toe.....	1	0	1
Fifth toe.....	1	0	4

Of all the fracture studies, 73.6 percent involved the bone at the tip of the great toe. Both bones of the great toe accounted for 82 percent of the fractures of all 14 bones of the toes.

FINANCIAL ASPECTS OF TOE INJURIES

In addition to the loss of productivity entailed in 1,153 sick days, the cash value of the lost time is \$7,300.49 for 1 year. This total is derived from the base per diem pay of each injured worker. The money loss for the 94 fractured toes was \$6,683.15, and the loss from the 19 severely contused toes was \$617.34.

During the period of this study the average number of employees (excepting clerks and draftsmen) was 10,845. The money loss from toe injuries per industrial employee was therefore 67.3 cents for the year.

The 437 riggers lost 140 days of working time with a pay value of \$783.74 or \$1.79 per man; almost three times the yard industrial average.

PREVENTION OF TOE INJURIES

The foregoing figures would have no point if toe injuries were not readily preventable. Most of this loss of \$7,300 in wages and 1,153 working days plus the expenditure of time and money for medical care is avoidable.

The most commonly used protective device is the safety shoe. This shoe has a metal toe-cap built into the shoe which completely covers and protects the bone at the tip of the great toe and the bones at the center and tip of the second toe. Seventy-one of our cases, or 7.5 percent, representing a time loss of 810 working days and \$5,162.66 in wages, had the injury fall entirely within this area.

There is abundant evidence that the metal tip of a safety shoe will protect the underlying toes against an impact of less than 300 foot pounds. Because the toe cap of the shoe rises higher than the vamp, it tends to break the force of blows that would otherwise threaten to fracture those portions of the toes not covered by the metal cap.

After careful consideration of the nature of the accident, and the type and location of injury in each of our lost-time cases, it is considered unlikely that more than seven fractures would have occurred if safety shoes had been worn.

Another commonly employed protective device is the foot guard. This is entirely independent of the shoe and is worn as an accessory. This covers a much greater area than the safety shoe, and will therefore protect the foot against fractures of the metatarsals as well as the toe bones. This protective device has only slight applicability to yard employees because of its bulk, and because climbing, stooping, and kneeling are practically impossible when the guard is worn. These objections do not apply to safety shoes.

It has been amply demonstrated in many industries that prevention of toe injuries by voluntary use of safety shoes is unsatisfactory. Personal observation has shown that practically none of the supervisors and leading men, and less than 5 percent of the industrial workers in this yard are wearing safety shoes at this time.

Case No. 3 of our series demonstrates how little may be accomplished by dependence on voluntary industrial safeguards. G. A., rigger, lost 4 working days in January 1931 because of a severe contusion of the toes of the left foot. In April 1937 he broke the toes of the same foot, required hospitalization, and lost 28 working days. In January 1940, still not wearing safety shoes, he again broke his left great toe.

The compulsory wearing of safety shoes by all industrial workers in this navy yard is the most practical single method of reducing the incidence of broken toes. Our figures indicate that this one measure promises to decrease the annual rate of accidents by approximately 10 percent.

A less efficient alternative method is the requirement of safety shoes only for workers engaged in the more hazardous occupations, such as riggers, sheet-metal workers, and laborers.

This alternative must not obscure the fact that the *only* way to eliminate the loss from broken toes is to make the wearing of safety shoes a requirement for all industrial workers, just as the use of welder's and chipper's goggles, leather protective clothing, ventilation blowers, and other safety measures have become an integral part of the working equipment in this yard.

RECOMMENDATIONS

(a) To make the wearing of safety shoes compulsory for all employees at this navy yard, with exception of draftsmen, and clerical and supervisory personnel.

(b) In the event that this measure cannot be applied to all workers, to make the wearing of safety shoes compulsory for riggers, sheet-metal workers, and laborers.

SUMMARY

1. There were 94 cases of fractured toes and 19 lost-time cases of contused toes among 10,846 industrial workers at this navy yard in 1 year.

2. The incidence of fractures was 0.83 percent, or 1 out of every 115 industrial workers. The combined fractures and contusions affected 1.04 percent, or 1 out of every 96 men.

3. There were 93 cases losing time due to toe injuries. This is 10 percent of lost time from all injuries for the year.

4. The time loss due to toe injuries was 1,158 days and the financial loss \$7,300.49. The average loss per man was 14.3 days. Toe injuries accounted for 12 percent of all time lost because of ordinary industrial accidents at this yard.

5. No cases occurred among clerks, draftsmen, or other officer personnel.

6. A marked difference in hazard existed among the 31 occupations involved in the 94 cases of fracture. Riggers represented 10.6 percent of the cases. One out of every thirty riggers had a broken toe during the year.

7. There is x-ray evidence that 75.5 percent of the cases would not have occurred if safety shoes had been worn. An additional 17 percent of the cases would probably have been prevented with this protection.

8. It is recommended that the wearing of safety shoes be made compulsory for all employees with exception of draftsmen, clerical, and supervisory personnel.

ACKNOWLEDGMENT

Grateful acknowledgment is made of the generous assistance of Capt. E. W. Brown, Medical Corps, United States Navy, in both the planning and the actual preparation of this report.

STATISTICS

HEALTH OF THE NAVY

The statistics (annual rates per 1,000) appearing in this summary were compiled from data contained in monthly reports of communicable diseases received in the Bureau for the months of October, November, and December 1940.

ENTIRE NAVY

Year	All diseases	Injuries and poisonings	All causes	Communicable diseases		Venereal diseases
				A	B	
1935-----	409	61	470	8	126	73
1936-----	474	76	550	15	118	96
1937-----	472	39	511	11	118	93
1938-----	325	57	382	4	76	81
1939-----	363	54	417	3	84	98
1940-----	511	49	560	35	223	81

FORCES ASHORE

1935-----	427	64	491	9	151	39
1936-----	459	98	557	13	121	46
1937-----	535	49	583	11	169	55
1938-----	330	55	386	7	104	40
1939-----	368	54	422	4	117	52
1940-----	487	49	536	27	229	52

FORCES AFLOAT

1935-----	344	59	457	8	111	93
1936-----	525	63	546	15	115	125
1937-----	398	34	469	10	89	116
1938-----	321	59	380	2	59	103
1939-----	360	54	414	3	64	126
1940-----	533	48	581	43	219	106

Common infectious diseases of the respiratory type.—During the quarter ending December 31, 1940, there were 14,515 admissions for these diseases reported for the entire Navy—7,596 from the forces afloat, 6,498 from shore stations in the United States, and 421 from foreign shore stations. Catarrhal fever was responsible for 11,582 of the admissions.

Ships and shore stations reporting the greatest number of admissions were as follows:

Ship or station	Octo-ber	Novem-ber	Decem-ber	Total
Naval Training Station, Great Lakes, Ill.....	179	224	756	1, 159
Marine Corps Base, San Diego, Calif.....	93	177	393	663
Naval Training Station, Newport, R. I.....	64	81	376	521
Naval Training Station, Norfolk, Va.....	143	102	229	474
U. S. S. <i>Enterprise</i>	280	29	77	386
U. S. S. <i>West Virginia</i>	131	97	65	293
U. S. S. <i>Colorado</i>	85	82	103	270
First Marine Brigade, Fleet Marine Force, Guan- tanamo Bay, Cuba.....	44	76	133	253
U. S. S. <i>California</i>	84	26	133	243
U. S. S. <i>Mississippi</i>	44	43	145	232
Naval Air Station, San Diego, Calif.....	40	135	34	209
U. S. S. <i>Idaho</i>	48	32	125	205
Naval Training Station, San Diego, Calif.....	48	31	122	201
Fleet Air Detachment, Battle Force.....	36	100	63	199
Receiving Ship, San Diego, Calif.....	5	58	123	186
Naval Air Station, Norfolk, Va.....	18	20	139	177
U. S. S. <i>Oklahoma</i>	58	90	24	172
U. S. S. <i>Pennsylvania</i>	5	75	92	172
U. S. S. <i>Tennessee</i>	96	11	63	170
U. S. S. <i>Arizona</i>	23	69	65	157
Navy Yard, Mare Island, Calif.....	5	34	116	155
U. S. S. <i>Saratoga</i>	39	29	76	144
U. S. S. <i>Nashville</i>	82	16	43	141
U. S. S. <i>Ranger</i>	114	8	17	139
Marine Barracks, Quantico, Va.....	16	43	77	136
U. S. S. <i>Nevada</i>	93	15	25	133
U. S. S. <i>Philadelphia</i>	62	17	53	132
U. S. S. <i>Savannah</i>	67	16	37	120
Receiving Ship, San Francisco, Calif.....	4	8	106	118
U. S. S. <i>San Francisco</i>	77	9	32	118
Naval Air Station, Pensacola, Fla.....	38	23	56	117
U. S. S. <i>Denebola</i>	21	19	71	111
Marine Barracks, Parris Island, S. C.....	30	34	40	104
U. S. S. <i>Lexington</i>	6	51	47	104
U. S. S. <i>New Mexico</i>	42	47	15	104
U. S. S. <i>Boise</i>	12	21	68	101
Naval Air Station, Seattle, Wash.....	9	16	72	97
Naval Air Station, Pearl Harbor, T. H.....	76	11	7	94
U. S. S. <i>Chaumont</i>	43	41	9	93
U. S. S. <i>Brooklyn</i>	10	11	70	91
Submarine Base, New London, Conn.....	10	10	70	90

In reporting the occurrence of respiratory diseases at various stations, the senior medical officers commented as follows:

Naval Training Station, Norfolk, Va. (special report dated December 21, 1940).—On December 21, with a personnel of 6,628, there were 242 men on the sick list, 162 of which had a diagnosis of catarrhal fever, acute. The disease was of a mild type necessitating the retention of the majority of the patients on the sick list from 4 to 5 days. Cases developing complications were transferred to hospital.

Naval Air Station, Pensacola, Fla. (sanitary report for October 1940).—There were 39 admissions to the sick list for diseases of the upper respiratory tract during the month of October, as follows:

1 case of angina, Vincent's; 28 cases of catarrhal fever, acute; 7 cases of tonsillitis, acute; and 1 case of pharyngitis, acute. All cases were of a minor nature, without complications.

Fleet Training Base, San Clemente Island (sanitary report for November 1940).—The general health of the crew has been excellent during the month with the exception of the last day, at which time there appeared an unusual number of complaints of "colds" and "flu" which were tentatively identified as influenza. Suspected individuals were promptly segregated and concurrent disinfection was practiced. All efforts were directed toward prevention of the disease in epidemic form.

A special epidemiological report dated December 4, 1940, stated that since November 30 there were 19 admissions with influenza, 6 of which were naval personnel and 13 W. P. A. personnel. Infected individuals were promptly isolated and the measures necessary to prevent the spread of the disease were instituted.

Naval Training Station, San Diego, Calif. (sanitary reports for November and December 1940).—Epidemic catarrhal fever is prevalent in San Diego. It appeared on this station early in November, averaging about 50 cases a day, totaling about 1,000 cases for the month. It is still present among the recruits, with a decreasing admission rate. Other units in San Diego are experiencing the present situation. The disease is mild, of 2 to 4 days' duration, affecting mostly the upper respiratory tract. Blood findings show little change in white and differential counts—no leukopenia and seldom a leukocytosis over 10,000. The disease is believed to be an epidemic form of catarrhal fever.

The sanitary report for the month of December stated that epidemic catarrhal fever or mild influenza continues. The character of the disease has changed. More cases show upper respiratory conditions with productive cough and sore throat. Acute tonsillitis with Vincent's angina is more prevalent. Many of the cases resemble the San Diego colds which are prevalent at this time of the year and which usually attack newcomers. All cases of a temporary nature are handled on the station.

Receiving Ship (U. S. S. "Rigel"), San Diego, Calif. (sanitary report for November 1940).—During the month of November there were 48 cases of catarrhal fever recorded, the first group of cases occurring on November 18. The prevalence of this disease is regarded to be in the form of a mild epidemic. The disease manifested itself in the form of light symptoms of three days' duration. No untoward effects were noted.

Receiving Ship, San Francisco, Calif. (sanitary report for November 1940).—On November 27, 1940, cases of mild influenza began to appear

at this station; there were 10 cases up to December 1. All cases showed elevation of temperature and mild respiratory symptoms. Local health officers report that mild influenza is quite prevalent in northern California.

The sanitary report for the month of December states that the epidemic of mild influenza which appeared on this station on November 27, reached its peak on December 6. A total of 140 cases were reported during a 17-day period. The infection was mild but of sufficient severity to require rest in bed from 4 to 7 days. There were no respiratory complications and no cases were transferred to hospital. Three cases developed acute otitis media.

Dispensary, Eleventh Naval District, San Diego, Calif. (sanitary report for December 1940).—Epidemic influenza continued during December with a marked increase in the number of admissions. Twenty-five cases were reported in November and 122 in December. Admissions in December were approximately 9.6 percent of the total complement. Every precaution was taken to treat and isolate the cases, and no doubt this contributed in some measure in keeping to a minimum the percentage affected. Most of the cases reported were of mild type; a few complications of severe bronchitis were noted.

Marine Corps Base, San Diego, Calif. (sanitary report for December 1940).—During the month a mild epidemic form of influenza (128 cases) and catarrhal fever (225 cases) occurred at this base. The cases were of a mild nature, without complications.

Headquarters, Twelfth Naval District, San Francisco, Calif. (sanitary report for December 1940).—A mild epidemic of influenza was prevalent during the month. Forty-eight cases of acute catarrhal fever, 1 case of acute laryngitis, and 2 cases of influenza were admitted during December. Mild complications were noted in a few cases.

Naval Air Station, Seattle, Wash. (sanitary report for November 1940).—An unusually high incidence of acute respiratory infections was noted during the final week of November. About 50 percent of the cases were mild influenza.

Headquarters, Thirteenth Naval District, Seattle, Wash. (sanitary report for December 1940).—An epidemic of influenza was experienced in this vicinity during the month. Twenty admissions with a total of 85 sick days were reported by this activity. There was 1 death due to this epidemic.

Marine Barracks, Naval Air Station, Kodiak, Alaska (sanitary report for December 1940).—During the period December 18 to December 31, 7 enlisted men were admitted with mild influenza, characterized in each instance by acute pharyngitis and low fever. Four of the cases had terminal hoarseness of a mild degree. The average number of sick days per case was 4.71. A large number of similar cases concurrently occurred in the local contractor's civilian personnel

and in the nearby village of Kodiak. This mild epidemic was undoubtedly transmitted from the Seattle area and according to present indications seems to be dissipating itself.

Naval Air Station, Pearl Harbor, T. H. (sanitary report for October 1940).—The admissions for catarrhal fever approached mild epidemic proportions in September and continued for the first 2 weeks in October, after which the admissions steadily declined.

U. S. S. Ranger (Guantanamo Bay, Cuba)—Special report dated October 29, 1940).—There were 105 cases of acute tonsillitis on board the *U. S. S. Ranger* from 1900 October 13, 1940, to 0800 October 17, 1940, as follows:

Date	Cases	Percent of total	Date	Cases	Percent of total
Oct. 13.....	51	48.57	Oct. 16.....	5	4.76
Oct. 14.....	33	31.43	Oct. 17.....	2	1.90
Oct. 15.....	14	13.33			

The distribution of cases was as follows:

6.77 percent of crew involved.

30 aviation personnel, or 28.57 percent of cases (comprise 32 percent of crew).

12 food handlers, or 11.43 percent of cases (comprise 8.06 percent of crew).

1 marine, or 0.95 percent of cases (comprise 5 percent of crew).

No officers involved.

No prisoners involved.

The cases presented a uniform picture of sudden onset, general malaise, elevated temperature, dysphagia, and tenderness of lymphatic gland of the neck. Lesion was characterized by a pseudo-membrane covering the crypts extending to varying degrees over the tonsils. Smear showed staphylococci and short chain streptococci with very few fusiform bacilli and *Spirochaeta vincenti*.

Lesion cleared:

First-second day..... 10 cases, or 9.52 percent.
 Second-third day..... 25 cases, or 23.8 percent.
 Third-fourth day..... 62 cases, or 59.02 percent.
 Fourth-fifth day..... 6 cases, or 5.71 percent.
 Fifth-sixth day..... 2 cases, or 1.90 percent.

Temperatures:

Normal..... 1 case, or 0.95 percent.
 99-100..... 44 cases, or 41.89 percent.
 100-101..... 30 cases, or 28.56 percent.
 101-102..... 13 cases, or 12.38 percent.
 102-103..... 12 cases, or 11.42 percent.
 103-104..... 3 cases, or 2.86 percent.
 104-105..... 2 cases, or 1.90 percent.

Duration of temperature :

12 to 24 hours.....	4 cases, or 3.81 percent.
24 to 48 hours.....	54 cases, or 51.41 percent.
48 to 72 hours.....	26 cases, or 24.75 percent.
72 to 96 hours.....	21 cases, or 19.99 percent.

The average isolation period was 5 days.

Measures to control epidemic were immediately instituted and consisted of :

(a) Sterilization of all scuttlebutts by the use of open flame. This was repeated twice daily during the epidemic.

(b) Constant supervision of scullery by pharmacist's mate during operation to insure proper sterilization of messgear.

(c) Immediate isolation of all hands with sore throats and fevers.

(d) Discontinued use of all coffee pots and cups about ship other than in galley.

(e) Closed ship's service soda fountain until all utensils in use had been checked and sterilized.

(f) Physical examination of all food handlers immediately and daily during epidemic.

(g) Notice placed in plan of the day concerning proper drinking habits and hygiene for scuttlebutts.

(h) Inspection of all sleeping spaces to insure proper spacing and positions.

(i) Word passed for all hands to report to sick bay if experienced sore throat.

(j) Telephone mouthpieces cleansed by alcohol after use.

(k) Galley and bake shop inspected thoroughly for foci.

(l) Specimen of water sent to hospital for examination and culture.

(m) Isolation wards established and proper signs posted.

(n) Separate mess and serving tables established in isolation areas. Separate messgear used.

ANALYSIS OF CASES IN GENERAL

(a) No conclusions could be drawn as to any scuttlebutt being involved; men had imbibed from every scuttlebutt on the ship.

(b) No conclusions could be drawn as to sleeping compartments; men involved came from all parts of the ship. The greatest frequency occurred in compartments where there was a greater concentration of men.

(c) No conclusion could be reached as to messing; both messes were involved and every table of both messes.

(d) Fifty-seven of the men involved, or 54.26 percent of the cases, had been ashore, so this was apparently not a factor.

(e) Twenty-eight of the men involved, or 26.64 percent of the cases, had used ship's service fountain; not considered foci.

(f) No cases of chronic tonsillitis were under treatment prior to epidemic, so carrier origin in question.

(g) Low and high temperatures preceding epidemic show :

Temperatures

Date	High	Low	Date	High	Low
Oct. 10.....	82. 5	73	Oct. 14.....	90	71
Oct. 11.....	81	72. 5	Oct. 15.....	90	70
Oct. 12.....	89	73	Oct. 16.....	89	70
Oct. 13.....	86. 5	72. 5	Oct. 17.....	90	72

The humidity was approximately 75.

These figures do not justify that a sudden change in temperature was the precipitating factor.

(h) A review of menus for the week preceding the epidemic revealed no article that could be implicated definitely.

(i) No milk was taken aboard after the ship left Norfolk, Va., on October 2, 1940; supply exhausted by October 5, 1940; unable to check on possibilities of milk as origin.

(j) Analysis of water by hospital essentially negative.

CONCLUSIONS

(a) The short duration of the epidemic justifies the opinion that proper measures were instituted for its control.

(b) The fact that no officers were involved limits the confines to the general mess and enlisted men's country but specific origin cannot fairly be stated.

U. S. S. Idaho (Hawaiian Area—Special report dated December 8, 1940).—The *U. S. S. Idaho* sailed from the Long Beach area on November 29, 1940, at 0900. At 0800 sick call 35 patients reported with acute follicular tonsillitis. Throughout that day 19 additional cases were admitted for the same condition, making a total of 54. Twenty-three cases were admitted on November 30 and 3 on December 1, or a grand total of 80 in 48 hours. Temperatures ranged from 99.0° F. to 102.6° F. on admission. Symptoms were sore throat, headache, and malaise. No new cases appeared after December 1.

Cases were isolated as they appeared. A moderately large athwartship berthing space just aft of the sick bay was cleared of personnel and their belongings. Forty-five of the most ill were put on cots and the rest used their hammocks in this space. Throat smears were made in each case. Treatment, T. P. R., hot saline gargle and 10

percent silvol swabs q. i. d. The cases were divided into 4 grades or types. Type I, some faucial inflammation with one or two scattered follicles; type II, considerable faucial inflammation with several follicles involved; type III, marked faucial inflammation, swelling and many infected follicles; and type IV, same as type III but with a false membrane. One half of each type was then put on sodium salicylate and sodium bicarbonate \overline{aa} grains XX, q. i. d., and the other half was put on sufamilamide grains, 80 the first day and 60 grains each day thereafter.

All throat smears showed many gram positive cocci, some single but the majority in short chains. The spirillum of Vincent and fusiform bacillus were also present but not in abnormal numbers. Identification of these cocci was not possible.

On December 2, cases which were symptom-free, whose tonsils had apparently returned to normal, and whose temperature had been normal for 48 hours were returned to duty. By December 4 all but 5 had returned to duty.

The source of the infection could not be determined but the cause of its rapid spread was undoubtedly due to overcrowding. Prior to November 1 the ship's complement was slightly less than 1,200. Washing, toilet, berthing, and messing facilities with this number was somewhat crowded. There were 1,154 hammock billets. By November 16 the number of crew on board was 1,862, of whom 278 were passengers. To accommodate the 400 increase in permanent complement, new hammock hooks were put up in every available space including the chief petty officers' mess hall, in passageways, and in offices. However, this made space for only 150 additional hammock billets. To accommodate the remaining men, 250 crew and 278 passengers, cots were crowded together under the hammocks, and in every available undercover deck space, and in offices. Mattresses were also used on office desks. This overcrowding meant that any semblance of sleeping head to foot which had been carried out fairly well with the 1,200 complement was impossible with 1,862.

Most of these men were aboard ship for the first time and from their appearance it is believed that many of them had been sick for 2 or 3 days and had not reported to the sick bay, either through ignorance or in fear of losing their liberty; the others apparently had had their resistance lowered by several late liberty parties. Most of the symptoms were far in excess of actual fever and throat condition and after 24 hours' rest in bed cleared up promptly.

The increase in personnel was made up entirely of men from training stations. It is interesting to note that 75 percent of the 80 cases were among nonrated men and 66 percent came from the

deck divisions, which were the most crowded. Interesting also is the fact that only three divisions did not have one or more cases. The H and N divisions are billeted entirely by themselves and the V division had been ashore for more than 3 weeks and came on board the night before sailing.

It is believed that the prompt isolation of all cases prevented the spread of what appeared to be a very serious epidemic, especially with the crowded conditions present.

Of note also is the fact that the sulfanilamide treatment did not return the men to duty as soon, or at least no sooner, than did the sodium salicylate-sodium bicarbonate treatment.

U. S. S. Cuyama (enroute San Pedro, Calif., special report dated October 23, 1940).—An epidemic of communicable diseases occurred on board this vessel during the 12-day period September 28 to October 10, 1940, as follows:

Date	New admissions	Total on sick report	Percent of complement on sick report
Sept. 28.....	2	2	1.3
Sept. 29.....	2	4	2.7
Sept. 30.....	0	4	2.7
Oct. 1.....	2	4	2.7
Oct. 2.....	8	12	8.0
Oct. 3.....	16	26	17.3
Oct. 4.....	7	29	19.3
Oct. 5.....	2	13	8.6
Oct. 6.....	2	12	8.0
Oct. 7.....	0	7	4.6
Oct. 8.....	2	4	2.7
Oct. 9.....	0	4	2.7
Oct. 10.....	0	1	2.7
Oct. 11.....	0	0	0.0
Total.....	43	43	28.6

Distribution by age groups

Age group	New admissions	Age group	New admissions
16-19.....	2	35-39.....	6
20-24.....	20	40-45.....	3
25-29.....	7		
30-34.....	5	Total.....	43

The average number of sick days per case was 2.8; shortest, 1 day; longest, 7 days. The average daily strength of crew was 150.

The first 4 cases were admitted with the diagnosis of catarrhal fever, but when the epidemic nature of the outbreak was recognized, subsequent cases were diagnosed as influenza. On October 3, 1940,

with 16 new admissions for influenza during the previous 24 hours, a modification of the ship's schedule was recommended in accordance with existing United States Fleet instructions. A delay of 24 hours in carrying out the scheduled sailing on October 4 was recommended, and after conference with the commander, base force, a delay of 48 hours was approved.

The crest of the epidemic was reached on October 4 when there were 7 new admissions, giving a total of 29, or 19.3 percent of the crew on the sick report.

All cases admitted presented practically the same symptoms—sudden onset, chilly sensations, fever, conjunctival injection, ocular soreness, headache, general pains, and constipation. The respiratory tract was involved in the majority of the cases. Physical examination was essentially negative with the exception of a mild conjunctival injection and a slight injection of the nasopharyngeal mucous membrane. No skin eruption was observed during the course of the disease.

A "saddle" type of temperature curve suggestive of dengue or denguelike infection was noted in seven of the cases. A considerable number of mosquitoes were in evidence in the navy yard area at Pearl Harbor during the stay of the U. S. S. *Cuyama*. None of the patients were hospitalized. However, the facilities of the sick bay (eight beds) soon became inadequate for the number requiring admission. Milder cases were cared for in the forward compartment which was fitted out as an accessory ward. The sick bay was reserved for those cases requiring more constant care. Blood counts were made on the severest cases and revealed a slight leukopenic tendency; otherwise, the counts were within normal limits.

The usual measures of control were instituted. Isolation of all cases was effected. Frequent checks were made on the scullery to see that all messgear was scrupulously cleaned and properly sterilized. Ventilation was maintained at a maximum throughout the ship. Decks of living spaces were swabbed down with compound cresol solution. The temperature of all men and officers was taken daily before breakfast and all personnel revealing any degree of morning temperature were admitted to the sick report. Food was prepared in the general mess and served the patients in messgear kept separate and sterilized after each meal. The program of motion pictures was curtailed during this period. After clearing Pearl Harbor, bedding was aired daily.

Complications.—The course was comparatively mild in all cases. The maximum temperature in the average case was 102.4° F. One patient developed an acute otitis media requiring myringotomy. There were no deaths.

It is believed that the disease was introduced on board in a mild unrecognized form simultaneously by several or more members of the crew or yard workmen, and that a large number of the crew had a milder form of the disease but did not consider themselves sick and therefore did not report to the medical officer or at the sick bay.

The explosive nature of the epidemic is shown by the fact that 31 (72 percent) of the 43 cases occurred during the 3-day period October 2 to October 4, 1940, inclusive, after which date the incidence rapidly declined. The first cases occurred among the engine room force.

Comment.—During an influenza epidemic aboard ship, the prompt isolation of all personnel with above-normal temperature is the earliest control measure to be taken. In addition to the desired isolation, this will put all cases to bed at the beginning of an attack, minimize the severity of the disease, and protect the patient from secondary infections.

U. S. S. Boise (en route Pearl Harbor, T. H.—Special report dated January 26, 1941).—During the period October 12 to December 31, 1940, inclusive, there were 4 officers and 82 enlisted men admitted to the sick list on board this vessel with the diagnosis of catarrhal fever, acute, and influenza, as follows:

Date	Catarrhal fever, acute	Influenza
Oct. 12-31.....	11	0
November.....	14	5
December.....	53	3
Total.....	78	8

Many other cases received ambulatory treatment. During December the sick bay was filled most of the time so that men with mild symptoms and temperatures below 99.6° F. were not admitted to sick list. Eleven bunks in an adjacent compartment were used to care for ward overflow. All of the cases were treated aboard ship.

The unusual increase in admissions dated from October 12. The ship had just returned to Long Beach, Calif., from the Hawaiian Islands, where the number of influenza cases was approaching epidemic proportions. The *U. S. S. Boise* departed from Long Beach on October 15 and after a stay of about a month in the Hawaiian Area returned to the West Coast, arriving in Mare Island, Calif., on November 22. Influenza cases were on a definite increase in California at this time and a slight increase in the number of cases of catarrhal

fever was noted aboard ship, accompanied by the admission of 5 cases diagnosed as influenza. In December there were 53 cases of catarrhal fever, acute, and 3 cases of influenza admitted to the sick list.

Although the number of cases increased markedly in December, the signs and symptoms of the average case remained mild. There were no deaths or serious complications. Two of the influenza cases showed signs of bronchial pneumonia. These cases were treated by administration of sulfapyridine and recovery was prompt. True influenza was confined to the enlisted personnel.

The treatment in all cases was that usually given to the ordinary case of catarrhal fever, acute, and proved to be adequate. The average duration of illness was from 4 to 5 days. Variations in treatment did not seem to affect this average. In only 2 cases was it thought necessary to give sulfapyridine.

The Battle Force Medical Bulletin dated October 24, 1940, reported:

An epidemic of mild influenza appeared throughout the Hawaiian Islands during the latter part of September. Average duration of illness was from 4 to 5 days with few complications being reported. Similar epidemics have occurred on several ships with from 10 to 30 percent of the personnel contracting the disease.

U. S. S. Enterprise (San Diego, Calif.—Special report dated November 23, 1940).—Epidemic influenza of mild intensity prevailed on board the *U. S. S. Enterprise* from September 23 to November 20, 1940.

Influenza was epidemic on the island of Oahu during this period and contact with the population was effected by week-end liberty parties.

The ship was actively employed with gunnery and aviation practices. Weather conditions were average.

Three hundred and thirty officers and men were admitted to the sick list. At the height of the epidemic 40 patients were transferred to the Naval Hospital, Pearl Harbor, T. H., in 3 days. One hundred cots were placed in a segregated area on the hangar deck.

Preventive measures included augmentation of the regular sanitary inspections. A patrol toured the ship every 4 hours, quizzed all men found lying about, and sent those not feeling fit to the sick bay for examination. All food handlers and the divisions showing the highest incidence in the preceding 24 hours were inspected each day. The master-at-arms force kept the living compartments clear during working hours and enforced head-to-foot sleeping regulations at night.

The character of average case was as follows: (a) Onset sudden with moderate prostration, temperature 101–103° F., disproportionately rapid pulse and respiration; (b) symptoms of headache, gen-

eralized rheumatoid pains, and slight dyspnoea; (c) physical examination essentially negative; (d) response to treatment satisfactory, usually afebrile in 3 days; (e) recurrence of signs and symptoms on fourth or fifth day not infrequent; (f) no significant complications; and (g) white blood counts on about 200 cases showed an average mild leukopenia.

The divisions most affected were the Marine Detachment (Fourth) "B," "M," and "S." The Marines supplied the majority of the admissions during the first week. They were mainly recruits, were berthed in a separate compartment, and had a marked tendency to congregate there when off duty. The admission rate was abnormally high due to the fact that daily medical inspections of personnel resulted in a number of mild or abortive cases being admitted to the sick list. The "B" and "M" divisions work in hot spaces and when they come off watch fall in on a hangar-deck chow line. This is a very draughty area when air operations are in progress, and it was noted that most of the admissions occurred during a period of air operations. The "S" division showed a parallelism between white and colored personnel. The daily inspection of food handlers undoubtedly increased the admission rate.

The divisions least affected were "A," "V-1," "V-2," "V-3," "N," and the signalman ratings of "K." These men are almost universally constant topside workers except the "A" division men, who are usually mature, higher rated men.

The air-group personnel showed a somewhat higher rate than the ship's air department personnel due in part to the fact that periods of great physical activity were followed by periods of idleness when the relatively overheated men remained on the flight deck where strong winds prevailed.

The distribution by rates were as follows: Officers, 2 percent; chief petty officers, 4 percent; petty officers, first class, 13 percent; petty officers, second class, 8 percent; petty officers, third class, 22 percent; and nonrated men, 50 percent.

U. S. S. Mississippi (Pearl Harbor, T. H.—Special report dated November 15, 1940).—A mild epidemic of acute catarrhal fever occurred on board this ship between November 20 and December 15, 1940. A total of 177 cases occurred between the above-mentioned dates. The majority of the 177 cases which occurred were returned to duty within 3 to 5 days. Forty-seven cases were on the sick list on December 5, the greatest number on the sick list at any one time.

One case of lobar and one case of broncho-pneumonia developed during the epidemic. These cases were treated on board with sulfapyridine until convalescence had started, when they were transferred to the *U. S. S. Relief*.

During the height of the epidemic, space in the "R" division compartment just aft of the sick bay was utilized as a ward and also space on the boat deck just beneath the signal bridge was rigged as a convalescent ward.

U. S. S. San Francisco (Pearl Harbor, T. H.—Special report dated November 1, 1940).—During the month of October 1940, 75 cases of catarrhal fever, acute, were admitted to the sick list aboard this vessel. At the same time this disease was present in epidemic form in Honolulu, T. H.

The *U. S. S. San Francisco* arrived in Pearl Harbor, T. H., on September 29, 1940. The first case was that of a mess attendant who had not been ashore or visited another ship. He was admitted to the sick list on October 3, 1940, and immediately isolated.

The following measures were instituted:

- (a) Water pressure was increased so that men would not be tempted to place their mouths on scuttlebutts.
- (b) Scuttlebutts were wiped with alcohol every 4 hours.
- (c) Extra care was taken in the sterilization of messgear.
- (d) Men were cautioned about common drinking and coffee cups and smoking others' partially used cigarettes.
- (e) Head-to-foot sleeping was instituted and inspections made.
- (f) Men were encouraged to sleep on deck when possible.
- (g) The hanging of wet clothing in sleeping compartments was forbidden.

The chief symptoms were high fever, frontal headache, pain in lumbar region, mild pharyngitis, and some prostration. White blood counts were made on a few cases and were normal or below normal. There were no complications and all cases were retained aboard ship.

On October 16 the disease assumed epidemic proportions and 15 cases were admitted to the sick list. The admiral's cabin and adjacent deck space were used as an auxiliary sick bay. Two mess cooks were detailed to the medical department. Separate messgear was used for patients and placed in boiling water for 5 minutes after being used.

A total of 75 cases occurred—2 officers and 73 enlisted men. There were 247 sick days recorded, or 3.28 sick days per case.

U. S. S. Salt Lake City (Pearl Harbor, T. H.—Special report dated November 2, 1940).—An epidemic of acute catarrhal fever occurred on board this vessel between the dates of September 23 and October 25, 1940.

The first wave of the epidemic was at its peak on October 5, 1940, when 10 cases were on the sick list. The epidemic subsided for a few days except for an occasional admission. On October 15 the second wave of the epidemic occurred, represented by 5 admissions. Control measures were immediately instituted. There was a total of

48 cases on board during the entire epidemic. The incidence of the infection was more or less equally distributed by divisions. The average duration of infection was 3 days.

Laboratory study as a whole showed normal to a slight reduction in the white blood cells, although there was an occasional patient with a white count of 10,000 to 12,000. This is in keeping with the diagnosis of influenza, but due to the mildness of the infection and because there were no complications, it was considered symptomatically acute catarrhal fever.

U. S. S. Pensacola (at sea, Hawaiian Area—Special report dated October 31, 1940).—A mild epidemic of catarrhal fever, acute, occurred on board the *U. S. S. Pensacola* during the latter part of September and the month of October 1940.

There were 7 cases of catarrhal fever, acute, on the sick list on September 27; and from that date to October 31, a total of 77 cases were admitted, 92 percent being admitted prior to October 16. It can be assumed, therefore, that the epidemic was definitely on the wane on October 16.

The daily average number of cases on the sick list was 6.9 from September 27 to October 16, and 1.0 from October 16 to October 30. The greatest number of cases for any one day was 16.

All cases presented exactly the same signs and symptoms. Leukopenia was not noted, the white counts of the more seriously ill patients being elevated with moderate shifts to the left. General malaise, headache, and slightly raw throat were the most common complaints. A large majority of the patients complained of lumbar backache on the second or third day of the disease. The average temperature upon admission was approximately 101.4° F. One definite recurrence (re-admission) was recorded. One case was complicated by pyelitis, acute.

The average number of sick days per case was 2.03. Forty-four of the seventy-seven cases admitted for treatment were returned to duty within 24 hours. One case was admitted from the Naval Ammunition Depot, Oahu, T. H., and transferred to hospital. The greatest number of sick days recorded for any one case was 6 days. The majority of the cases occurred among the deck divisions, as the following table indicates:

Deck force.....	41
Engineer force.....	15
Marines.....	12
Special branch.....	8
Officers.....	1
Total.....	77

It was noted that the members of the deck force responded slightly more rapidly and readily to treatment than the special branch or

engineer force. The average age of the deck force was considerably below that of the engineer force.

Immediately upon realization that a mild epidemic was in progress the following measures were instituted:

- (a) The men were cautioned to avoid crowded places while ashore and to avoid large gatherings in compartments aboard ship.
- (b) The entire crew was made cognizant of the disease and encouraged to report to the sick bay with the slightest headache, sore throat, generalized aching, etc.
- (c) Scullery, galleys, and scuttlebutts were checked frequently.
- (d) Well-defined cases were promptly isolated.
- (e) Ventilation was checked in sleeping compartments and men encouraged to sleep in the open with necessary precautions.
- (f) Difficulty was met during this epidemic in obtaining good ventilation due to normal operation of the ship, including damage-control practice runs.

The important etiological factors were personal contact, exposure, exhaustion, and loss of sleep.

U. S. S. Kane (San Francisco, Calif.—Special report dated December 19, 1940).—Mild cases of catarrhal fever, acute, began to appear on board this vessel on December 5, 1940, and 15 cases were admitted to the sick list between this date and December 19.

The cases were of mild nature with elevation of temperature and mild respiratory symptoms. There were no pulmonary complications and no cases were transferred to hospital.

Other infectious diseases.—Cases of measles, German measles, and mumps were reported for October, November, and December, as follows:

Ship or station	Measles	German measles	Mumps
U. S. S. <i>Argonne</i>	0	2	0
U. S. S. <i>Arizona</i>	1	11	1
U. S. S. <i>Arkansas</i>	0	4	0
Fleet Air Detachment, Battle Force.....	0	2	0
U. S. S. <i>Brooklyn</i>	0	8	0
U. S. S. <i>California</i>	5	1	0
U. S. S. <i>Capella</i>	1	0	0
U. S. S. <i>Chaumont</i>	0	0	2
U. S. S. <i>Chicago</i>	4	1	1
U. S. S. <i>Colorado</i>	1	37	2
U. S. S. <i>Denebola</i>	0	2	10
U. S. S. <i>Dixie</i>	0	1	0
U. S. S. <i>Enterprise</i>	2	1	1
U. S. S. <i>Holland</i>	0	1	0
U. S. S. <i>Idaho</i>	11	1	32

Ship or station	Measles	German measles	Mumps
U. S. S. <i>Indianapolis</i>	0	1	0
U. S. S. <i>Lexington</i>	1	35	0
U. S. S. <i>Louisville</i>	0	3	0
U. S. S. <i>Maryland</i>	6	18	0
U. S. S. <i>Medusa</i>	1	0	0
U. S. S. <i>Melville</i>	0	2	0
U. S. S. <i>Milwaukee</i>	0	1	0
U. S. S. <i>Minneapolis</i>	0	4	0
U. S. S. <i>Mississippi</i>	38	4	0
U. S. S. <i>Nevada</i>	3	1	0
U. S. S. <i>New Mexico</i>	0	21	0
U. S. S. <i>Northampton</i>	0	2	0
U. S. S. <i>Oglala</i>	1	0	0
U. S. S. <i>Oklahoma</i>	0	1	2
U. S. S. <i>Omaha</i>	0	1	0
U. S. S. <i>Pennsylvania</i>	0	16	5
U. S. S. <i>Phoenix</i>	0	0	1
U. S. S. <i>Portland</i>	1	0	0
U. S. S. <i>Quincy</i>	0	0	2
U. S. S. <i>Raleigh</i>	0	6	0
U. S. S. <i>Relief</i> (duty personnel).....	0	7	0
U. S. S. <i>Richmond</i>	1	0	1
U. S. S. <i>Salt Lake City</i>	0	0	1
U. S. S. <i>San Francisco</i>	0	3	0
U. S. S. <i>Saratoga</i>	3	8	2
U. S. S. <i>Savannah</i>	0	4	0
U. S. S. <i>St. Louis</i>	0	7	2
U. S. S. <i>Tennessee</i>	0	9	0
U. S. S. <i>Texas</i>	0	2	0
U. S. S. <i>Tuscaloosa</i>	5	0	17
U. S. S. <i>Vincennes</i>	0	5	0
U. S. S. <i>West Virginia</i>	2	22	1
U. S. S. <i>Whitney</i>	0	3	1
U. S. S. <i>Wyoming</i>	0	2	1
Navy Yard, Boston, Mass.....	1	0	0
Submarine Base, New London, Conn.....	1	1	1
Naval Air Station, Lakehurst, N. J.....	2	0	0
Naval Air Station, Anacostia, D. C.....	0	0	1
Naval Academy, Annapolis, Md. (midshipmen).....	3	18	5
Naval Academy, Annapolis, Md. (others).....	0	1	1
Naval Proving Grounds, Dahlgren, Va.....	1	0	0
Naval Air Station, Norfolk, Va.....	0	8	4
Naval Training Station, Norfolk, Va.....	8	76	25
Norfolk Naval Hospital, Portsmouth, Va.....	3	1	0
Norfolk Navy Yard, Portsmouth, Va.....	0	1	0
Navy Yard, Charleston, S. C.....	0	0	1
Marine Barracks, Parris Island, S. C.....	0	0	2
Naval Air Station, Jacksonville, Fla.....	0	0	1
Naval Station, Key West, Fla.....	0	1	0
Naval Hospital, Great Lakes, Ill.....	0	0	1
Naval Training Station, Great Lakes, Ill.....	0	0	1
Marine Scouting Sqd. 3, FMF, St. Thomas, V. I.....	1	0	0
Marine Corps Base, San Diego, Calif.....	1	0	4
Naval Air Station, San Diego, Calif.....	2	1	0
Naval Hospital, San Diego, Calif.....	0	2	0
Hospital Corps School, San Diego, Calif.....	0	2	0
Naval Training Station, San Diego, Calif.....	0	17	4
Receiving Ship, San Diego, Calif.....	0	7	0
Naval Air Station, Terminal Is., San Pedro, Calif.....	3	0	0
Navy Yard, Mare Island, Calif.....	1	0	1
Puget Sound Naval Hospital, Bremerton, Wash.....	1	0	0
Puget Sound Navy Yard, Bremerton, Wash.....	1	0	0
Naval Air Station, Seattle, Wash.....	0	3	1

Ship or station	Measles	German measles	Mumps
Naval Air Station, Pearl Harbor, T. H.-----	3	0	0
Naval Hospital, Pearl Harbor, T. H.-----	0	1	0
Navy Yard, Cavite, P. I.-----	0	1	0
4th. Marines, Shanghai, China-----	6	0	0
Marine Detachment, Tientsin, China-----	0	0	1
1st. Marine Brig. FMF, Guantanamo Bay, Cuba--	0	1	0
Naval Station, Guantanamo Bay, Cuba-----	1	0	0
Naval Station, Guam, M. I.-----	0	0	1

Septic sore throat—*U. S. S. "California"* (en route Long Beach, Calif.—Special report dated December 27, 1940).—While operating in the Hawaiian area in November 1940, an outbreak of 152 cases of sore throat of a clinically septic type occurred on board and subsided in 8 days. The outbreak was limited to enlisted personnel and distributed throughout the ship. Admissions were recorded as follows: First day, 116; second day, 19; third day, 6; fourth day, 4; fifth day, 3; sixth day, 0; seventh day, 3; and eighth day, 1. There were no deaths, serious complications, or transfers to other activities for treatment.

The chief complaint was sore throat. Other symptoms were malaise, headache, backache, occasional muscle and joint pains, and fever. The tonsils were the primary site of invasion in every case except those in which tonsillectomy had been done. In the early stage the appearance of the tonsils was that of an acute follicular tonsillitis; the tonsils were infected, slightly swollen, and spotted with a grayish-white exudate. The tonsillar pillars and soft palate were also involved and presented a fairly marked infection. Within a few hours the tonsils became markedly swollen and the follicular exudate increased and coalesced to form a dirty grayish, foul-smelling, tenacious, necrotic exudate. The reaction of the contiguous soft structures was severe and consistent with the progress of the infection in the tonsils and small, yellowish, shallow ulcers were occasionally found on the pillars, soft palate, and pharynx. In patients whose tonsils had been removed the signs and symptoms were milder except in the occasional case where remnants of tonsillar tissue were present. At onset the temperature ranged from normal to 104° F. and was consistent with the degree of involvement of the throat. Fever and local symptoms subsided in from 2 to 7 days although in cases with markedly cryptic tonsils, swelling and edema persisted for several days beyond the febrile period.

Throat smears were stained by Gram's method and were negative for diphtheria in every case. The usual field showed Gram positive organisms in the form of long and short chains and cocci diffusely

scattered or in clusters. Ten swabs were made from throats of the earlier cases and sent to the Naval Hospital, Pearl Harbor, T. H., for culture for diphtheria and hemolytic streptococcus. All were reported negative for diphtheria. Five were positive for the hemolytic streptococcus, 1 for the nonhemolytic streptococcus, and 4 were negative for these organisms.

Treatment consisted of strict isolation of the patient and his mess-gear, dietetic measures, aspirin and bicarbonate of soda, and hot saline gargles. Sulfanilamide was used only in cases with peritonsillar complication. Patients remained in isolation and under treatment until all signs of the membranous exudate and ulceration had disappeared and the temperature had been normal for 3 days. Making cultures of the throat and establishing special isolated messes for patients discharged from treatment were considered not to be practical or necessary. Peritonsillar abscess complicated 17 cases, 9 of whom required surgical drainage, 3 drained spontaneously, and 5 responded to sulfanilamide. One case was complicated by a painful swollen wrist which quickly responded to sulfanilamide only to recur in an exaggerated and resistant form 72 hours later.

Control measures were directed toward the detection and elimination of a human source of infection and the medium of transmission of the disease from person to person. The examination of all food handlers and a careful inspection of their throats was immediately carried out and repeated daily until the outbreak had subsided. The only food handler among the first several admissions was a ship's cook's striker, although four ship's cooks were admitted within the first 12 hours. The latter were examined and their throats inspected 8 hours before reporting for treatment. Evidence of throat involvement or constitutional symptoms were not apparent on the first examination but when admitted all of the signs and symptoms of a moderately severe acute tonsillitis were present. Messgear contaminated by an infected person or persons was immediately suspected as being the medium of transmission. The identity of the human source could not be positively established, as the mechanical dishwasher and sterilizer had long been completely out of commission. Replacement had been authorized during the navy yard overhaul the previous summer, but certain obstacles arose to prevent installation. In 1939, when the operating efficiency of the plant became questionable, sterilization of clean messgear was attempted by dipping in a tank of boiling water for 1 minute, the tank being specially installed for this purpose. An outbreak of acute tonsillitis and an epidemic of acute catarrhal fever following the adoption of this plan demonstrates the inadequacy and danger of such makeshift arrangement,

and the present outbreak of septic sore throat is unquestionably believed to be due to a large number of persons eating and drinking from contaminated messgear. The paramount control measure, therefore, was to insure complete sterilization of all eating and drinking utensils used by the general mess and careful attention to the sterilization of all gear used in officers' and other messes. The bedding and clothing steam sterilizer on the boat deck was surveyed as to its usefulness for this purpose. An effective plan was developed by bypassing the steam jacket and supplying live steam directly to the chamber with pressure controlled by a gage specially installed for this purpose. This plan was carried out for 8 weeks and proved its efficiency by promptly controlling the current epidemic and resulting in a marked reduction in admissions for upper respiratory diseases. Fortunately, a contemplated availability period at a navy yard insures the installation of a new mechanical dishwasher and sterilizer.

From the foregoing it is believed reasonable to conclude that contaminated messgear was the medium of transmission of a virulent, epidemic type of sore throat which was promptly controlled by measures insuring proper sterilization. The relative decrease in the admission rate for communicable diseases of the respiratory type serves to further confirm this conclusion.

Chickenpox.—Seven cases of chickenpox were reported for the quarter—two from the U. S. S. *Tennessee* and one each from the U. S. S. *Trenton*, Marine Barracks, Parris Island, S. C., Naval Training Station, Great Lakes, Ill., Marine Corps Base, San Diego, Calif., and Naval Training Station, San Diego, Calif.

Scarlet fever.—Single cases of scarlet fever were reported in October from the U. S. S. *Altair* and the U. S. S. *Trenton*, and in December from the Naval Training Station, Norfolk, Va.

Diphtheria.—The Marine Detachment, Peiping, China, reported a case of diphtheria in November.

Cerebrospinal fever, meningococcic.—An apprentice seaman, 20 years of age, with 3 months' service at the Hospital Corps School, Norfolk, Va., was admitted to the sick list with catarrhal fever, acute, on October 16, 1940, and transferred to the Norfolk Naval Hospital the same day, where the diagnosis was changed to cerebrospinal fever, meningococcic. He was returned to duty on November 22.

An apprentice seaman, 18 years of age, with 26 days' service, was admitted to the Naval Training Station, Newport, R. I., with "Diagnosis undetermined" and transferred to hospital on November 19, 1940. The diagnosis was established as cerebrospinal fever, meningococcic. The patient was discharged to duty on January 24, 1941.

Meningitis, cerebrospinal, acute.—An apprentice seaman, 21 years of age, with 3 months' service, was admitted to the sick list on board the U. S. S. *St. Louis* on December 11 with meningitis, cerebrospinal, acute, and discharged to duty December 23, 1940.

Typhus fever.—Three cases of moderately severe uncomplicated typhus fever were admitted to the Regimental Hospital, Fourth Marines, Shanghai, China, in November and 1 case to the Naval Hospital, Parris Island, S. C., in October 1940.

Gastroenteritis, acute, Naval Training Station, Great Lakes, Ill. (special report dated December 10, 1940).—On Saturday afternoon, November 16, 1940, at about 1550, 75 patients reported to the dispensaries, showing typical signs and symptoms of gastroenteritis, acute. The patients were immediately transferred to the hospital and remained on the sick list for 3 days. These patients, without exception, came from Camp Barry and were served the same noon meal as was served at the other mess halls on the station. Inasmuch as there were no complaints from other activities, it is assumed the cause was in the preparation or handling of the food, rather than in the food.

The suspected menu consisted of navy bean soup, boiled ham and cabbage, boiled potatoes, sour pickles, jello, bread, butter, and coffee. The ham was boiled the previous night, not chilled, and allowed to stand until 0400, November 16, at which time it was sliced for the noon meal. Laboratory examinations:

Ham: Gram positive cocci arranged in clusters.

Impression: *Staphylococcus albus* and gram negative bacilli.

Feces: Gram positive cocci and gram negative bacilli.

Impression: *Staphylococcus albus*.

Vomit: Gram positive cocci.

Impression: *Staphylococcus albus* and *staphylococcus aureus*.

Several sources of contamination are possible, although it is thought the poisoning was due to bacterial contamination of a small amount of ham, inasmuch as only about 5 percent of the men who ate this meal were affected. The galley in question (Camp Barry) has been under a long siege of repair, in and near the area of food preparation, repair work being carried on by civilians from various localities, who may have been the source of contamination.

FOOD POISONING

Fourth Defense Battalion, Fleet Marine Force, Parris Island, S. C. (special report dated December 20, 1940).—There were 111 cases of food poisoning at this post on Saturday, December 14, 1940.

At 1130 on the above date 2 men came to the sick bay complaining

of violent nausea and epigastric pain. Before anything could be done for them, 6 more cases appeared, and in a few minutes 20 or 30 more. All cases were immediately transported to the naval hospital of this post and given emergency treatment, consisting of gastric lavage, morphine sulphate, and in some cases strychnine or adrenalin chloride. Intravenous saline was given to 5 cases for severe muscular cramps and dehydration.

The outbreak was explosive in nature and involved 111 men, all of whom, except one or two, were affected between the hours of 1000 and 1400. Questioning revealed that all affected men had eaten egg spread for breakfast and none had eaten since that time. The eggs had been boiled at 1730 the previous night, part of them peeled, and all of them placed in the refrigerator. At 0330 on December 14 the remaining eggs were peeled and all were chopped and mixed with dressing, made of butter, salad oil, milk, flour, water, salt and pepper, and Worcestershire sauce, spread on toast and served for breakfast. A sample of this was obtained from the side of a garbage can, and the sample submitted to the laboratory at the hospital. Samples of vomitus and feces were also submitted. The laboratory findings from vomitus and excreta were disappointing, but a growth, identified as belonging to the *Salmonella* group, was obtained from the food specimen.

DETAILS

Symptoms.—Onset was sudden, from $\frac{1}{2}$ hour to 7 hours after eating, with the peak from 5 to 6 hours after breakfast, characterized by uneasiness, anorexia, nausea, vomiting, epigastric cramps, diarrhea, and abdominal cramps. Most patients were quite pale, very weak, and bathed in cold sweat. Some developed muscular cramps from chloride loss and dehydration. Symptoms lasted until good gastric lavage was obtained and gradually subsided in about 1 to 2 hours, although many were feeling weak and had no appetite the following morning. All but three were able to perform duty within 48 hours.

Temperature.—Ranged from 97° F. to 98.8° F. and many of the patients complained of feeling cold. One chill was noted.

Urine.—Normal.

Blood.—White count ranged between 6,000 and 32,000 with a mean of 12,000.

Vomit.—Contained undigested particles of egg. No case showed bloody vomitus.

Cramps.—All cases were violently nauseated and complained of epigastric pain. Many complained of abdominal cramps.

Diarrhea.—Was common, but not as prominent a feature as nausea.

Eyes.—There were no ocular symptoms.

Treatment.—Gastric lavage; bed rest; preservation of body heat; strychnine sulphate $\frac{1}{60}$ grain or adrenalin 1:1000 minims 3 for stimulation; morphine sulphate grains $\frac{1}{8}$ for cramps and to control diarrhea; and in those cases in which it was indicated, intravenous saline solution gave much relief from cramps and dehydration.

The cases which were prostrated were kept in the hospital until sufficiently recovered to be returned to the barracks. All of the cases were returned to the barracks by the morning of the next day.

From a consideration of the facts it is believed that this outbreak was due to egg spread infected by a bacterium of the Salmonella group, probably coming from the hands of the cook. This was made possible by improper preparation and handling of food and overtaxation of messing facilities.

Fifth Defense Battalion, Fleet Marine Force, Marine Barracks, Parris Island, S. C. (sanitary report, December 1940).—An epidemic of food poisoning occurred in this battalion on Saturday December 14, 1940, due to bacteria enteritidis. The outbreak was explosive in nature and involved 77 men. All cases were immediately transported to the naval hospital and given emergency treatment consisting of gastric lavage, morphine sulphate, and in some cases strychnine or adrenalin chloride. Intravenous saline was given to three cases for severe muscular cramps and dehydration.

Symptoms.—Onset was sudden, from $\frac{1}{2}$ hour to 7 hours after eating, with the peak from 5 to 6 hours after eating breakfast, characterized by uneasiness, anorexia, nausea, vomiting, epigastric cramps, diarrhea, and abdominal cramps. Most of the patients were quite pale, very weak, and bathed in cold sweat. The men were able to perform duty after 48 hours.

Breakfast consisted of chopped eggs mixed with dressing made of butter, salad oil, milk, flour, water, salt, pepper, and Worcestershire sauce, spread on toast. Specimens of food, vomitus, and feces were submitted to the laboratory and the findings supported the diagnosis of bacteria enteritidis. A growth identified as belonging to the Salmonella group was obtained from the food specimen from the mess, probably coming from one of the food handlers. Temperature was normal, urine normal, and white count ranged from 6,000 to 32,000. Vomitus contained undigested particles of eggs. No cases showed bloody vomitus. All cases were violently nauseated and diarrhea was common. No cases required admission to the sick list.

U. S. Naval Air Station, Pensacola, Fla. (sanitary report, October 1940).—There was an epidemic of food infection involving 43 men on October 31. The infection was traced to ham served at the noon meal on the general mess. The first indication of illness appeared about $3\frac{1}{2}$ hours after eating. The poisoning affected a group served

approximately at the same time, indicating that one or two hams were infected.

The symptoms were sudden onset with vomiting and intestinal cramps accompanied by diarrhea.

Laboratory tests made at the United States Naval Hospital, Pensacola, Fla., on the gastric contents and from samples of ham showed straphylococcus albus in pure culture. Twenty-one patients were hospitalized. The remainder were sent to their barracks because of mild symptoms and were fit for duty within 24 hours.

First Battalion, Fifth Regiment, First Marine Brigade, Fleet Marine Force, Guantanamo Bay, Cuba (annual sanitary report, 1940).—There was an outbreak of food poisoning among the personnel of the First Battalion, Fifth Regiment, on October 31, 1940. The outbreak was attributed to ground boiled ham sandwiches which were prepared in the following manner: The hams were boiled from 1900 to 2300 on the preceding day, placed in a field ice chest for 4 hours, ground, mixed with mayonnaise and pickle, and made into sandwiches. They were dispensed at 0600 and carried in field haversacks in the hot sun for 6 hours before they were eaten. Forty-five of the four hundred men who ate these sandwiches were admitted to the sick list, 19 of them being severely ill. Several other men reported to the sick bay with mild symptoms but were not admitted to the sick list. Bacteriological examination of the sandwiches revealed a heavy contamination with a highly motile, Gram negative bacillus of the colon group. Further differentiation was not possible.

The feeding of men in the field in the manner described above is ideal for the development of such an outbreak of food poisoning.

CADMIUM POISONING

First Marine Aircraft Group, Fleet Marine Force, Guantanamo Bay, Cuba (annual sanitary report, First Marine Brigade, 1940).—On October 20, 1940, 31 men reported to sick bay with the following symptoms of acute poisoning: Projectile vomiting, abdominal cramps, and diarrhea. In the majority of cases symptoms appeared within 2 hours following the early mess at 1100. All affected cases drank lemonade which came from an aluminum 10-gallon container, and complained of the extremely sour taste of the beverage. Investigation disclosed that the following meal was served: Fried pork chops, boiled potatoes, buttered peas, bread and butter, and lemonade. All men affected ate early mess at 1100 and consumed lemonade dispensed from a particular aluminum container. This was the only difference in their food consumption when compared with the

men eating at late mess, who did not become ill. The lemonade for the noon mess was prepared in the following manner:

- (a) Preparation began at 1030 and consumption completed at about 1200 on October 20.
- (b) A 10-gallon aluminum container was used and not washed out prior to use.
- (c) Unwashed lemons were cut with rinds and placed in another metal container and juice extracted with a wooden masher.
- (d) The juice and rinds were divided into three portions, one portion being placed in the container in question.
- (e) Sugar, ice, and water were added for the completion of the beverage. The ice was from Government inspected and approved sources and was from the same supply used in the other two containers for the late mess, which was consumed without ill effects.
- (f) A regulation metal dipper was used for the dispensing.

The patients were admitted to the sick list and received treatment at the field dispensary and in their respective living tents. Routine treatment consisted of 1 dram each of paregoric and bismuth subcarbonate every 3 hours. The majority of cases recovered from acute symptoms within 4 hours after onset. All of the cases made uneventful recovery and returned to duty within 3 days.

Conclusion.—The only common item consumed by those affected was the lemonade dispensed from one particular 10-gallon aluminum container. Barring unknown accident or willful contamination, the causative agent is believed to be chemical, namely* aluminum citrate, formed by the action of lemon juice (citric acid) on the aluminum container. The container in question was confiscated for inspection. All of the contents, however, had been used and the container rinsed out, making it impossible to make any chemical analysis of the responsible beverage.

Recommendations.—

- (a) No acid foods or beverages be placed in the new 10-gallon aluminum food containers.
- (b) Strict compliance with sanitary regulations pertaining to thorough cleansing of all containers.
- (c) Washing of lemons prior to use. Juice to be extracted mechanically and rinds discarded.
- (d) Strict enforcement of regulations prohibiting unauthorized personnel in galleys to avoid possibility of accidental or malicious food and beverage contamination.

*See footnote on page 410, showing poisoning due to cadmium.

MORBIDITY

Summary for the quarter ending December 31, 1940

TABLE 1.—Total, United States Navy

	Forces afloat		Forces ashore		Entire Navy	
Average strength	126,806		112,973		239,779	
	Admis- sions	Rate per 1,000	Admis- sions	Rate per 1,000	Admis- sions	Rate per 1,000
All causes	18, 430	581. 36	15, 129	535. 67	33, 559	559. 83
Diseases only	16, 895	532. 94	13, 759	487. 16	30, 654	511. 37
Injuries and poison- ings	1, 535	48. 42	1, 370	48. 51	2, 905	48. 46
Communicable dis- eases transmissible by oral and nasal discharges (class VIII):						
(A)	1, 366	43. 09	752	26. 63	2, 118	35. 33
(B)	6, 937	218. 82	6, 456	228. 59	13, 393	223. 42
Venereal diseases	3, 354	105. 80	1, 474	52. 19	4, 828	80. 54

TABLE 2.—Summary, Naval districts, for the quarter ending Dec. 31, 1940

District	Average strength	Annual rates per 1,000			
		All causes	Diseases only	Respiratory diseases	Venereal diseases
First naval district	6, 655	619. 08	571. 60	383. 47	11. 42
Third naval district	5, 854	504. 27	439. 36	139. 39	45. 78
Fourth naval district	3, 905	442. 51	402. 56	170. 04	55. 31
Activities on the Severn and Potomac Rivers	13, 590	376. 75	321. 71	104. 49	39. 15
Fifth naval district	14, 958	514. 77	473. 59	202. 97	67. 39
Sixth naval district	6, 821	416. 36	382. 35	116. 70	38. 12
Seventh naval district	1, 754	410. 49	353. 48	155. 07	61. 57
Eighth naval district	5, 346	362. 14	316. 50	102. 51	47. 89
Ninth naval district	6, 855	987. 89	956. 96	738. 73	17. 51
Tenth naval district	724	657. 46	591. 16	82. 87	243. 09
Eleventh naval district	25, 736	435. 19	405. 66	252. 72	31. 86
Twelfth naval district	3, 786	767. 04	730. 06	573. 69	27. 47
Thirteenth naval district	3, 621	487. 16	442. 97	267. 33	39. 77
Fourteenth naval district	5, 827	330. 19	287. 63	140. 72	24. 03
Fifteenth naval district	2, 335	474. 52	417. 99	99. 36	92. 51
Sixteenth naval district	1, 442	457. 70	421. 64	47. 16	127. 60
Outlying stations	7, 573	870. 46	746. 86	222. 37	167. 44

TABLE 3.—*Venereal diseases (class XII) and communicable diseases transmissible by oral and nasal discharges (class VIII), ships and shore stations, for the quarter ending Dec. 31, 1940*

	Venereal diseases		Communicable diseases	
	Rate per 1,000, fourth quarter, 1940	Median rate, fourth quarter, 1935-39	Rate per 1,000, fourth quarter, 1940	Median rate, fourth quarter, 1935-39
SHIP				
Altair.....	87.69	92.11	94.44	50.85
Argonne.....	61.88	64.60	286.92	25.00
Arizona.....	55.84	53.48	502.57	114.08
Arkansas.....	85.74	127.78	221.73	60.77
Astoria.....	25.19	153.85	130.98	100.59
Augusta.....	536.53	327.72	143.71	92.44
Barnett.....	92.93	¹ 9/25/40	149.49	¹ 9/25/40
Battle Force, Fleet Air Detachment.....	38.07	31.03	225.08	64.76
Black Hawk.....	397.63	363.64	148.37	133.55
Boise.....	66.67	¹ 8/12/38	396.08	¹ 8/12/38
Brooklyn.....	143.34	¹ 9/30/37	443.45	¹ 9/30/37
California.....	52.49	73.21	1,102.21	94.09
Canopus.....	410.76	270.99	92.91	92.54
Chaumont.....	140.97	170.64	569.75	63.64
Chester.....	106.46	68.75	182.51	30.67
Chicago.....	22.32	73.20	267.86	62.75
Cincinnati.....	143.59	90.29	123.08	28.64
Colorado.....	78.97	106.81	846.83	137.12
Concord.....	138.55	80.65	271.08	43.48
Curtiss.....	212.23	¹ 11/15/40	293.86	¹ 11/15/40
Denebola.....	158.05	¹ 1/16/40	759.88	¹ 1/16/40
Detroit.....	24.00	55.85	40.00	64.37
Dixie.....	85.88	¹ 4/25/40	122.03	¹ 4/25/40
Dobbin.....	16.44	68.87	65.76	92.39
Enterprise.....	115.52	¹ 5/12/38	¹ 1,229.24	¹ 5/12/38
Helena.....	53.55	¹ 9/18/39	82.39	¹ 9/18/39
Henderson.....	110.64	182.78	85.11	97.73
Holland.....	93.82	99.81	255.86	71.71
Honolulu.....	98.58	¹ 6/15/38	125.12	¹ 6/15/38
Houston.....	479.50	63.49	113.56	78.18
Idaho.....	83.64	89.53	627.31	86.10
Illinois.....	6.86	¹ 8/1/40	343.05	¹ 8/1/40
Indianapolis.....	67.65	88.47	321.35	44.03
Langley.....	457.33	78.05	135.27	136.59
Lexington.....	67.92	101.48	330.21	115.57
Louisville.....	501.24	89.60	258.06	121.99
Marblehead.....	314.85	206.76	193.20	68.82
Maryland.....	47.75	62.94	283.82	113.30
McCawley.....	122.62	¹ 8/6/40	84.57	¹ 8/6/40
Medusa.....	44.26	89.14	348.55	60.36
Melville.....	153.31	47.71	299.65	78.74
Memphis.....	90.35	85.44	73.92	62.62
Milwaukee.....	162.71	78.59	325.42	47.15
Minneapolis.....	37.87	90.91	326.63	147.52
Mississippi.....	127.88	70.37	731.46	86.67
Nashville.....	133.48	¹ 6/6/38	627.36	¹ 6/6/38
Nevada.....	64.83	55.92	444.08	81.36
New Mexico.....	77.24	136.32	294.91	115.11
New Orleans.....	20.41	84.08	239.80	131.34
New York.....	149.07	114.29	62.11	58.44
Northampton.....	24.78	51.87	312.27	63.40
Oklahoma.....	56.70	70.05	453.61	53.64
Omaha.....	233.33	143.37	213.33	51.47
Pennsylvania.....	66.81	62.94	566.45	70.50
Pensacola.....	22.25	90.47	255.91	51.70
Philadelphia.....	140.43	¹ 9/23/37	561.70	¹ 9/23/37
Phoenix.....	52.86	¹ 10/3/38	185.02	¹ 10/3/38
Portland.....	74.26	95.24	445.54	71.92
Prairie.....	125.73	¹ 8/5/40	237.49	¹ 8/5/40
Quincy.....	111.76	¹ 6/9/36	274.74	¹ 6/9/36
Raleigh.....	62.89	71.11	572.33	117.04
Ranger.....	69.63	79.70	333.73	117.65
Relief (duty personnel).....	65.81	64.86	438.76	124.61
Richmond.....	160.00	60.61	153.60	17.32
Salt Lake City.....	62.83	76.19	282.72	63.49
San Francisco.....	53.08	75.58	583.84	96.90
Saratoga.....	99.89	93.86	337.94	101.44
Savannah.....	68.03	¹ 3/10/38	566.89	¹ 3/10/38
St. Louis.....	164.38	¹ 5/19/39	155.95	¹ 5/19/39
Tennessee.....	75.68	106.71	489.19	140.60
Texas.....	75.69	65.04	221.25	63.53

¹ Commissioned.

TABLE 3.—*Venereal diseases (class XII) and communicable diseases transmissible by oral and nasal discharges (class VIII), ships and shore stations, for the quarter ending Dec. 31, 1940—Continued*

	Venereal diseases		Communicable diseases	
	Rate per 1,000, fourth quarter, 1940	Median rate, fourth quarter, 1935-39	Rate per 1,000, fourth quarter, 1940	Median rate, fourth quarter, 1935-39
SHIP—continued				
Trenton.....	139.30	100.63	384.74	69.10
Tuscaloosa.....	172.25	87.08	239.23	63.86
Utah.....	0	103.90	291.75	129.68
Vestal.....	80.15	65.19	160.29	91.39
Vincennes.....	186.53	1 2/24/37	191.71	1 2/24/37
Wasp.....	100.29	1 4/25/40	61.95	1 4/25/40
West Virginia.....	71.11	81.33	807.62	91.92
Whitney.....	78.67	93.46	381.24	85.47
Wichita.....	84.12	1 2/16/39	126.18	1 2/16/39
Wyoming.....	111.11	150.94	200.00	82.33
Yorktown.....	77.94	1 9/30/37	87.99	1 9/30/37
STATION				
Naval Air Station, Anacostia, D. C.	18.96	11.98	75.83	18.60
Naval Academy (midshipmen), Annapolis, Md.	0	1.74	174.32	191.34
Naval Academy (other than midshipmen), Annapolis, Md.	157.04	63.13	117.01	86.42
Marine Barracks, Quantico, Va.	45.30	35.19	121.37	101.13
Marine Barracks, Washington, D. C.	22.54	23.32	157.75	108.84
Dispensary, Washington, D. C.	0	(2)	146.91	(2)
Navy Yard, Washington, D. C.	42.15	28.08	35.13	41.50
Navy Yard, Boston, Mass.	14.67	10.10	127.14	101.01
Naval Torpedo Station, Newport, R. I.	7.56	16.70	52.93	29.93
Naval Training Station, Newport, R. I.	9.22	5.07	604.03	417.63
Navy Yard, Portsmouth, N. H.	7.60	18.22	60.84	136.67
Naval Hospital, Brooklyn, N. Y.	55.85	(2)	160.56	(2)
Submarine Base, New London, Conn.	45.21	30.37	186.73	99.78
District Headquarters, New York, N. Y.	0	0	6.91	13.61
Navy Yard, New York, N. Y.	45.25	24.69	99.55	98.77
Receiving Ship, New York	91.95	95.76	76.63	76.63
Naval Air Station, Lakehurst, N. J.	37.04	31.25	129.63	78.43
Naval Hospital, Philadelphia, Pa.	19.56	(2)	391.20	(2)
Navy Yard, Philadelphia, Pa.	35.00	18.66	160.98	65.24
Receiving Station, Philadelphia, Pa.	114.38	62.02	165.21	51.63
Naval Air Station, Norfolk, Va.	60.48	64.00	308.95	104.00
Naval Training Station, Norfolk, Va.	61.34	62.27	248.31	303.93
Receiving Station, Norfolk, Va.	122.96	74.53	88.38	55.36
Norfolk Naval Hospital, Portsmouth, Va.	20.89	(2)	365.54	(2)
Norfolk Navy Yard, Portsmouth, Va.	98.84	55.34	154.44	68.52
Navy Yard, Charleston, S. C.	63.27	45.45	133.57	54.79
Fourth Defense Battalion, Parris Island, S. C.	58.78	(2)	117.56	(2)
Fifth Defense Battalion, Parris Island, S. C.	59.19	(2)	443.90	(2)
Marine Barracks, Parris Island, S. C.	27.40	(2)	97.74	(2)
Naval Air Station, Jacksonville, Fla.	66.99	(2)	210.53	(2)
Naval Station, Key West, Fla.	73.13	(2)	102.38	(2)
Naval Air Station, Miami, Fla.	63.16	(2)	112.28	(2)
Naval Air Station, Pensacola, Fla.	50.51	55.14	97.76	79.63
Reserve Midshipmen's School, Northwestern University, Chicago, Ill.	0	(2)	416.53	(2)
Naval Reserve Signal School, Chicago, Ill.	39.22	(2)	186.27	(2)
Naval Hospital, Great Lakes, Ill.	0	(2)	196.38	(2)
Naval Training Station, Great Lakes, Ill.	21.33	14.39	954.06	132.22
Naval Air Station, San Juan, P. R.	279.07	(2)	107.33	(2)
Dispensary, San Diego, Calif.	0	0	183.49	14.23
Marine Corps Base, San Diego, Calif.	36.22	33.02	211.34	114.89
Naval Air Station, San Diego, Calif.	25.45	25.29	359.63	50.82
Naval Hospital, San Diego, Calif.	16.00	(2)	648.00	(2)
Naval Training Station, San Diego, Calif.	26.21	12.28	173.44	226.24
Receiving Ship, San Diego, Calif.	42.32	47.31	281.65	28.25
Navy Yard, Mare Island, Calif.	15.77	37.15	495.27	25.87
Receiving Ship, San Francisco, Calif.	72.45	45.07	610.61	29.74
Puget Sound, Navy Yard, Bremerton, Wash.	34.84	39.54	160.28	27.83
Naval Air Station, Seattle, Wash.	66.61	29.37	288.66	64.61
Third Defense Battalion, Pearl Harbor, T. H.	10.24	(2)	158.77	(2)
Naval Air Station, Pearl Harbor, T. H.	17.54	34.19	130.90	82.05
Navy Yard, Pearl Harbor, T. H.	30.30	21.98	147.19	11.98
Submarine Base, Pearl Harbor, T. H.	44.35	61.70	177.38	123.39
Naval Air Station, Coco Solo, C. Z.	60.17	71.23	71.63	86.25
Submarine Base, Coco Solo, C. Z.	163.42	112.00	225.68	143.22

¹ Commissioned.

² Not available.

TABLE 3.—Venereal diseases (class XII) and communicable diseases transmissible by oral and nasal discharges (class VIII), ships and shore stations, for the quarter ending Dec. 31, 1940—Continued

	Venereal diseases		Communicable diseases	
	Rate per 1,000, fourth quarter, 1940	Median rate, fourth quarter, 1935-39	Rate per 1,000, fourth quarter, 1940	Median rate, fourth quarter, 1935-39
STATION—continued				
Navy Yard, Cavite, P. I.	159.09	144.14	22.73	56.34
Marine Detachment, Peiping, China	325.93	112.50	118.52	392.86
Fourth Marines, Shanghai, China	454.15	209.15	272.49	171.28
Marine Detachment, Tientsin, China	355.56	(2)	266.67	(2)
Marine Barracks, Guam, Marianas Islands	132.45	80.00	211.92	59.26
Naval Hospital, Guam, Marianas Islands	45.45	(2)	0	(2)
Naval Station, Guam, Marianas Islands	19.05	18.60	152.38	40.68
First Marine Brigade, Guantanamo Bay, Cuba	97.14	(2)	233.68	(2)
Mobile Base Hospital, Guantanamo Bay, Cuba	54.05	(2)	198.20	(2)
Naval Station, Guantanamo Bay, Cuba	159.31	100.00	278.81	52.06
Naval Station, Tutuila, Samoa	20.62	0	144.33	118.34

² Not available.

DEATHS

During the quarter ending Dec. 31, 1940

Cause		Number	Navy		Marine Corps		Nurse Corps	Midshipmen
Primary	Secondary		Officers	Men	Officers	Men		
Average strength		239, 779	14, 983	179, 119	2, 261	40, 320	500	2, 596
<i>Diseases</i>								
Abscess, amebic, liver	Mediastinitis	1		1				
Appendicitis, acute	Peritonitis, general, acute	3		2		1		
Appendicitis, acute	Pneumonia, lobar, type unknown	1		1				
Calculus, kidney	Obstruction, intestinal, from spastic or paralytic causes	1	1					
Carcinoma, stomach, type unknown		1		1				
Coronary heart disease, arteriosclerotic		3	2	1				
Encephalitis, acute		1		1				
Fistula, gastro-jejuno-colic		1		1				
Gangrene, colon	Thrombosis, inferior mesenteric vein	1		1				
Heart disease, congenital (anomaly, aortic valves)	Endocarditis, subacute, bacterial	1		1				
Hemorrhage, cerebral		1		1				
Do	Arteriosclerosis, general	1		1				
Hypertensive heart disease		2	1	1				
Influenza	Encephalitis, acute	1		1				
Do	Pneumonia, lobar	1		1				
Do	Toxemia, bacterial (organism unspecified)	1	1					
Malaria, malignant tertian	Pneumonia, broncho	1				1		
Melanoma, back		1		1				
Meningitis, cerebrospinal, acute		1		1				
Nephritis, chronic	Myocarditis, chronic	2	1	1				
Obstruction, intestinal, from external causes		1				1		
Pneumonia, broncho	Tonsillitis, acute	1		1				
Pneumonia, lobar		1	1					
Psychosis, exhaustive		1		1				
Septicemia, staphylococcus aureus		1		1				
Thrombosis, coronary artery		2		2				

DEATHS—Continued

During the quarter ending Dec. 31, 1940—Continued

Cause		Number	Navy		Marine Corps		Nurse Corps	Midshipmen
Primary	Secondary		Officers	Men	Officers	Men		
<i>Diseases—Continued</i>								
Tonsillitis, acute	Abscess, multiple	1		1				
Tuberculosis, pulmonary, chronic, active, far advanced		1						
Tumor, mixed malignant, teratoma, testicle		2		2				
Ulcer, duodenum	Hemorrhage, duodenum	1	1					
Ulcer, duodenum, perforated	Peritonitis, general, acute	1		1				
Total for diseases		39	8	27	0	4	0	0
<i>Injuries and poisonings</i>								
Burn, multiple		2	1	1				
Crush, chest		1		1				
Drowning		4		3			1	
Do	Fracture, compound, skull	2	1	1				
Do	Fracture, simple, skull	1	1					
Exhaustion from overexertion	Dementia praecox	1				1		
Fracture, compound, frontal		1		1				
Fracture, compound, skull		16	3	12		1		
Do	Meningitis, cerebrospinal, acute	1		1				
Fracture, simple, vertebra, cervical	Intraspinal injury	2		2				
Fracture, simple, skull	Intracranial injury	1				1		
Injuries, multiple, extreme		22	4	12	4	2		
Injuries, multiple, extreme	Psychosis, unclassified	1		1				
Intracranial injury		4		2		2		
Do	Psychosis, unclassified	1		1				
Rupture, traumatic, kidney		1				1		
Strangulation, respiratory		1		1				
Wound, gunshot, abdomen		1			1			
Wound, gunshot, head		4	1	3				
Do	Psychoneurosis, anxiety neurosis	1	1					
Wound, gunshot, heart		1		1				
Poisoning, acute, potassium cyanide		2		1		1		
Poisoning, therapeutic, acute, sulfapyridine (Urethritis nonvenereal)	Granulocytopenia, malignant	1		1				
Total for injuries and poisonings		72	12	45	5	9	1	0
Grand total		111	20	72	5	13	1	0
Death rate per 1,000 (annual basis):								
All causes		1.85	5.34	1.61	8.85	1.29	8.00	
Diseases only		0.65	2.14	0.60		0.40		
Drownings		0.12	0.53	0.02			8.00	
Poisonings		0.05		0.04		0.10		
Other injuries		1.03	2.67	0.94	8.85	0.79		

MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

Statistics for the quarter ending December 31, 1940

The following statistics were taken from sanitary reports submitted by naval training stations.

October, November, and December, 1940	United States naval training station			
	Norfolk, Va.	Newport, R. I.	Great Lakes, Ill.	San Diego, Calif.
Recruits received during the period.....	5, 676	5, 027	7, 311	6, 565
Recruits appearing before Board of Medical Survey.....	100	41	15	48
Recruits recommended for discharge from the service.....	86	29	29	45
Recruits discharged by reason of medical survey.....	66	(1)	10	(1)
Recruits held over pending further observation.....	39	14	(1)	0
Recruits transferred to the hospital for treatment, operation, or further observation for conditions existing prior to enlistment.....	0	41	(1)	73

¹ Not reported.

The following table was prepared from reports of medical surveys in which disabilities or disease causing the survey were noted existing prior to enlistment. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office.

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Abscess, periapical.....	1	Color blindness.....	2
Absence, acquired, teeth.....	5	Constitutional psychopathic inferiority without psychosis.....	14
Adhesions, abdominal.....	1	Constitutional psychopathic state, criminalism.....	1
Albuminuria.....	2	Constitutional psychopathic state, emotional instability.....	15
Amblyopia.....	1	Constitutional psychopathic state, inadequate personality.....	9
Amnesia.....	1	Constitutional psychopathic state, sexual psychopathy.....	1
Arthritis, chronic.....	3	Cryptorchidism.....	2
Asthma.....	7	Curvature, spine.....	2
Astigmatism, compound, hyperopic.....	2	Cyst.....	1
Astigmatism, simple, hyperopic.....	1	Cyst, teratoma, inflamed.....	1
Bronchitis, chronic.....	2	Deafness, bilateral.....	4
Bronchiectasis.....	1	Deformity, acquired.....	6
Bursitis, chronic.....	1	Deformity, congenital.....	5
Cardiac arrhythmia, auricular, extra systoles.....	1	Dementia praecox.....	22
Cardiac arrhythmia, premature contractions.....	2	Deviation, nasal septum.....	1
Cardiac disorder functional.....	2	Dislocation, articular cartilage.....	1
Caries, teeth.....	3		
Cicatrix, skin.....	1		

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Dislocation, chronic	6	Psychoneurosis, hysteria	12
Dislocation, lens	1	Psychoneurosis, neurasthenia	2
Eczema	1	Psychoneurosis, psychasthenia	1
Effort syndrome	3	Psychoneurosis, situational	1
Enuresis	35	Psychoneurosis, traumatic	2
Epilepsy	40	Psychoneurosis, unclassified	1
Flat foot	34	Psychosis, with other disabilities (mental deficiency)	1
Fracture, simple	1	Psychosis, unclassified	1
Gastritis, chronic	1	Ptosis, eyelid, congenital	1
Gonococcus infection, urethra	5	Rheumatism, muscular	1
Hammer toe	1	Rheumatic fever	2
Hernia, epigastric	1	Seasickness	1
Hernia, inguinal, indirect	20	Sinusitis, ethmoidal	1
Hernia, muscle	1	Sinusitis, frontal	1
Hydrocele, tunica vaginalis	1	Sinusitis, maxillary	3
Hydronephrosis	1	Situs, viscerum inversus	1
Hypertension, arterial	9	Somnambulism	9
Hypertrophy, ligamentum flavum	2	Spur bone	1
Ichthyosis	1	Stammering	1
Insufficiency ocular muscle	1	Strabismus	2
Joint, internal derangement of	3	Strain, muscular	1
Loose body in joint	1	Synovitis, chronic	1
Loss of substance, bone or cartilage	1	Syphilis	7
Malocclusion, teeth	5	Tuberculosis, adenitis, cervical	1
Mental deficiency (moron)	5	Tuberculosis, pulmonary, chronic, arrested, moderately advanced	2
Migraine	6	Ulcer, duodenum	5
Myopia	21	Ulcer, stomach	1
Nephritis, chronic	10	Union of fracture, faulty	4
Neuritis, multiple	1	Valvular heart disease, aortic and mitral	3
Neurosis, intestinal	1	Valvular heart disease, mitral insufficiency	7
Osgood-Schlatter disease	1	Valvular heart disease, mitral stenosis	2
Osteoarthropathy, hypertrophic	1	Vertigo	1
Osteomyelitis	1	Wart	1
Otitis media, chronic	28		
Otitis, externa	1		
Pansinusitis	2		
Paralysis, nerve	1		
Paradentosis	3		
Periostitis, chronic	1		
Pes cavus	1	Total	465

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FOR THE INFORMATION OF
THE MEDICAL DEPARTMENT OF THE NAVY



DIVISION OF PUBLICATIONS
THE BUREAU OF MEDICINE AND SURGERY



THE MISSION OF THE MEDICAL DEPARTMENT OF THE NAVY

•
TO KEEP AS MANY MEN AT AS MANY GUNS AS
MANY DAYS AS POSSIBLE



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NAVY DEPARTMENT,
Washington, March 20, 1907.

This UNITED STATES NAVAL MEDICAL BULLETIN is published by direction of the Department for the timely information of the Medical and Hospital Corps of the Navy.

TRUMAN H. NEWBERRY,
Acting Secretary.

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PREFACE

THE UNITED STATES NAVAL MEDICAL BULLETIN was first issued in April 1907 as a means for supplying medical officers of the United States Navy with information regarding the advances which are continually being made in the medical sciences, and as a medium for the publication of accounts of special researches, observations, or experiences of individual medical officers.

It is the aim of the Bureau of Medicine and Surgery to furnish in each issue special articles relating to naval medicine, descriptions of suggested devices, clinical notes on interesting cases, editorial comment on current medical literature of special professional interest to Medical Department personnel, and reports from various sources, notes, and comments on topics of professional interest.

The Bureau extends an invitation to all medical and dental officers to prepare and forward, with a view to publication, contributions on subjects of professional interest.

In order that each service contributor may receive due credit for his efforts in preparing matter for the BULLETIN of distinct originality and special merit, the Surgeon General of the Navy will send a letter of appreciation to authors of papers of outstanding merit.

The Bureau does not necessarily undertake to endorse views or opinions which may be expressed in the pages of this publication.

ROSS T. MCINTIRE,
Surgeon General, United States Navy.

VII

NOTICE TO CONTRIBUTORS

Contributions to the **BULLETIN** should be typewritten, doubled spaced, on plain paper and should have wide margins. Fasteners which will not tear the paper when removed should be used. Nothing should be written in the manuscript which is not intended for publication. For example, addresses, dates, etc., not a part of the article, require deletion by the editor. The **BULLETIN** endeavors to follow a uniform style in heading and captions, and the editor can be spared much time and trouble, and unnecessary changes in manuscript can be obviated if authors will follow in these particulars the practice of recent issues.

The greatest accuracy and fullness should be employed in all citations, as it has sometimes been necessary to decline articles otherwise desirable because it was impossible for the editor to understand or verify references, quotations, etc. The frequency of gross errors in orthography in many contributions is conclusive evidence that authors often fail to read over their manuscripts after they have been typewritten.

Contributions must be received at least 3 months prior to the date of the issue for which they are intended.

The editor is not responsible for the safe return of manuscripts and pictures. All materials supplied for illustrations, if not original, should be accompanied by reference to the source and a statement as to whether or not reproduction has been authorized.

The **BULLETIN** intends to print only original articles, translations, in whole or in part, reviews, and reports and notices of Government or departmental activities, official announcements, etc. All original contributions are accepted on the assumption that they have not appeared previously and are not to be reprinted elsewhere without an understanding to that effect and that editorial privilege is granted to this Bureau in preparing all material submitted for publication.

LOUIS H. RODDIS, *Editor*,
Captain, Medical Corps, United States Navy.
HILTON W. ROSE, *Assistant Editor*,
Lieutenant Commander, Medical Corps,
United States Naval Reserve.

U. S. NAVAL MEDICAL BULLETIN

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SPECIAL ARTICLES

FOREWORD BY THE SURGEON GENERAL OF THE NAVY

Rear Admiral Ross T. McIntire, Medical Corps, United States Navy

Although a carefully selected physical group, the health of the officers of the Navy may be adversely affected due to features of their occupation. The two most important of these are: (1) the heavy load of responsibility which most officers carry and which is particularly heavy in these trying times; (2) the lack of opportunity for recreation and exercise due to the pressure of official duties.

The Secretary of the Navy has taken cognizance of these features and in a communication addressed to the whole Navy (Alnav. 100, dated October 18, 1940) he has urged the importance of exercise and recreation as health measures.

It is in connection with the subject of exercise and recreation that Lieutenant Commander Tunney has emphasized this need to maintain the physical well-being of all personnel, particularly by means of appropriate exercise. I commend to all readers of the NAVAL MEDICAL BULLETIN his article which was prepared and delivered to midshipmen at the United States Naval Academy. It is deserving, however, of the attention of not only our future officers, but all members of the Navy.

The paragraph in Notes and Comments "Physical Fitness of Officers" on page 571 should also be read. This note draws special attention to the necessity that exercise must be individual in character to prevent its being detrimental, and must be suited to the age and physical condition of the person.

PHYSICAL FITNESS OF NAVAL PERSONNEL¹

By Lieutenant Commander J. J. Tunney, United States Naval Reserve

As you know I am here to talk on your physical fitness program. Our Secretary, Mr. Knox, issued on October 18th an order called ALNAV 100. ALNAV 100, in case you don't recall it, reads as follows:

Modern war requires the acme of physical fitness and fighting edge. Nothing shall be left undone to insure that all officers and men of our Navy are properly conditioned to meet the utmost demands of physical endurance. Paragraph 8 of General Order 122 must be considered a minimum requirement. All commands

¹ Address delivered to the Officers, Midshipmen, and Enlisted men at the United States Naval Academy, Annapolis, Maryland, May 26, 1941.

afloat and ashore will immediately institute adequate and systematic exercises to bring personnel to peak of physical fitness. Such measures will contribute to the continuance of the present high morale of the Navy.

General Order 122 reads, in part, as follows:

The value of taking regularly some form of physical exercise in the open air for at least 30 minutes each day is recognized. Commanding officers are urged to encourage officers to engage in sport and in regular exercise. In this connection daily setting-up exercises which maintain correct posture and muscular tone especially in the abdominal region, are recommended.

In connection with that, I think it oftentimes happens that service personnel take orders of this kind very lightly. A great many men who have spent a good deal of their time in the Navy, either as enlisted men or officers, get to the point where they feel that they know all naval customs, all regulations, and are willing to do anything within reason; but some of them feel, after so long a time, they are qualified to put their own interpretation on orders, which frequently results in neglecting the orders. Well, in speaking of that, there is a little message on discipline by Marshal Foch which I think should be appropriate, and is in line with that kind of reasoning. It is this:

To be disciplined does not mean either that one executes orders received only in such measure as seems proper or possible, but it means that one enters freely into the thought and aims of the chief who has ordered, and that one takes every possible means to satisfy him.

The first condition to obeying is, therefore, to visualize all the order received and nothing else, then to find the means of complying with it, irrespective of personal opinions, difficulties or obstacles.

Now, gentlemen, that is exactly the way the Bureau of Navigation feels about ALNAV 100; and there are many reasons why it feels that ALNAV 100 should be obeyed to the fullest by all men afloat and ashore.

For many, many centuries there has been the problem of staying physically fit for both military men and civilians. Military men have felt it obligatory. It is a serious problem because when men approach middle age they start to get that middle-age flabbiness of muscle, particularly those muscles that have to do with posture. I contend that "good posture is good health." Without good posture there cannot be good health permanently. So the first thing that one must do to be assured of good health is to acquire good posture. From a personal point of view that is important. From an organization point of view for the military man it is necessary.

Those officers who go through the Academy go through a very rigorous routine. They are required to have good health and an excellent heart; otherwise they couldn't finish. When they graduate they are in excellent condition with a fine, strong, muscular heart. It is what they do after they leave the Academy, when they have

reached the peak of perfection—physical perfection—that causes them either to stand properly at attention, when out a few years, or not to; also, either to get obese or to get too thin. It all has to do with their physical habits after graduating.

When you go through a routine every day for 4 years with some vigorous form of exercise you develop what is sometimes called an "athletic heart." For the want of a better name it is called simply that, but in truth it is an excellent heart; it is a strong muscular heart that is built up to stand a hard, vigorous grind. When you have developed the so-called athletic heart it requires thereafter a tribute every day; otherwise, like ordinary muscles, it will get flabby; and in that process of flabbiness come cardiac troubles. Many of our outstanding athletes have developed a heart thrombosis. Sometimes it causes their death. Sometimes it does not do the job completely, but leaves them invalids. And this is all due, in my opinion, or at least a great deal of it is due, to the fact that they do not pay to the heart that tribute which the heart requires after having been built up to the strength that enables it to absorb all the punishment occasioned by the constant exercise of the Academy routine.

The United States Navy is, without question, the finest organization of engineers and administrators in the world. The United States Navy, as you know without my telling you, is the finest Navy in the world. And to have so fine a Navy they have naturally had to develop excellent administrators and engineers. It is all right for an organization to look like engineers, also to look like administrators and act like administrators, but where that organization is a military organization, then the least you can expect of each member of that organization is that he looks military; and that they, collectively and individually, look like military people. There is an obligation on the part of those administrators and engineers not only to be great engineers and administrators but also to be and look like military men. And that is the important purpose of our physical-fitness program. It is to make members of the United States Navy look like military people because they are, first, military people, then they are administrators and engineers. If we are to look like military people, we have to start very early. When a man leaves this Academy he looks like a military man, but after he has been out a while he can look just the opposite. And so it is with the enlisted men. When an enlisted man first comes into the Service he has to go through months of training and he often does look the part of a military man; but after a while he gets sluggish and careless about his military appearance. Of course, his uniform is in order and he looks spick and span from that angle, but as a military man, one who is physically fit, he is on the whole, I would say, disappointing. So we have to think of this physical-fitness pro-

gram from the point of view of where it is going to take us with reference to the military appearance of the enlisted man as well as the officer.

There is a tendency after early youth for muscles to stretch, and there is a particular tendency for abdominal muscles to stretch. And the tendency is more developed in those who don't wear belts or suspenders but have to hold up heavy trousers by pushing their bellies out against the belt line of the trousers. That develops an elongation of muscles. The particular muscles that I am talking about are the girdle muscles coming around from their attachment at the spine to front-center of the stomach where they join. Those muscles have a tendency to relax and lengthen which brings on the "pot-belly." In my opinion, it is the stretching of these muscles—these girdle muscles—that causes 98 percent of the officers and enlisted men of some years service to have big stomachs. There are exercises that are called corrective exercises that one must do to keep those stomach muscles firm and short. When they are kept firm and short a man stands erect, but when they are allowed to relax from either too much sitting, or too much pushing against trousers, a man develops a sag; and when he stands at attention after 4 or 5 years during which he does his physical drills but once a week the chances are that he will not know how to assume the correct posture. He stands carelessly, with his back arched in, his hands hanging forward of his legs, his stomach out, and his chin sagging. His shoulders may be up, but because his shoulders are erect he thinks he is at attention. The correct position of attention is to have a vertical stance—a straight line from head to foot—so that a huge skewer pushed through would find center without curves.

But there is no question that one's stomach must be drawn in, the diaphragm drawn up, the head erect, the chin in. When you do that your chest comes out. After a while standing that way becomes normal, but if we allow our natural tendency to develop we will be like the fellow who goes to the tropics to live. I have known several of them. One is a great friend of mine. The first unusual thing to happen is he goes a couple of days without shaving. After a while he's got a beard. Then he decides that his shoes are too much trouble to put on, they will only wear away, so he doesn't put his shoes on. After this he thinks that any underclothing is bad, so he gets hold of a pair of shorts somewhere, puts them on and gets by without a shirt. He's got a beard now, doesn't have to wash, has bare feet, wears shorts without a shirt. Then he decides that getting up in the morning is too much trouble and he stays in bed. Then his rum is served in bed. He just follows his natural tendency. And the natural tendency of a human being is laziness. There is no question about that. No one likes to get up in the morning. No one

likes to climb upstairs. It's natural to fall into the philosophy of laziness. We must train ourselves against the tendency to drop our stomachs when standing up. Make a conscious effort. After a while you look and act like military men if you consider these matters and take the necessary corrective measures.

There are a number of exercises that we could take for corrective measures. I am going through these exercises to show you they are simple and won't take long to do. If you midshipmen, after you get out of the Academy, will remember some of them, and if you will remember to pay that tribute once a day to your heart, you will always be fit for rigorous experience. Because one in the Navy doesn't know when he will be called upon to stand long watch or to go through a terrifically exacting experience physically. If he has kept up his exercises, paid the little tribute each day, his heart tone is strong. Men who are in the Navy can get only irregular opportunity to play the games that give them exercise. Those who do this and nothing more are likely to develop heart conditions earlier than they should. That intermittent exercise is the worst kind of exercise unless you are supplementing it daily with other exercises. To play tennis every day for weeks straight and then stop for months, in which we overfeed, participate in no games or exercise, and then come back and play every day for a week or two, is the worst possible thing you can do for your health. But, if you, every morning, before breakfast, will do just 8 or 10 minutes of setting-up exercises and then have your shower, you will handle successfully those intermittent periods of intensive exercise and other experiences of unexpected physical exertion that come in line of duty. You will stand it because your heart has been attuned to it—because your heart has a good healthy tone. If you will remember and treat the heart as you would a muscle you will go through life without curtailing its functioning unexpectedly, through lack of the proper respect for it. Treat your heart as a friend and remember it commands respect; remember also that you have got to pay that tribute one day in the form of some physical exertion, no matter how slight.

I remember, at the age of 15 or 16, reading that Jess Willard daily put his feet under a piano, sat on a stool, and went back with his hands behind his head until he touched the floor, returning until his forehead touched his knees, 50 times. I started to do that. At the age of 31 I stopped, after 15 years of daily application. I developed a great wall of abdominal muscles but it looked as though I had a potbelly. I remember once a great friend of mine saying to me when I was training for the first Dempsey match in 1926, "Why you can't be in shape, you have a potbelly." I actually had a potbelly because I was stretching these muscles, these abdominal muscles. Instead of concave I was making them convex.

These are all facts I am speaking to you about, learned and observed from a lot of experience and also a great deal of study during, and subsequent to, my boxing career. These exercises, called "Suction Correctives," that I am about to give, were designed for the purpose of shortening abdominal muscles. They are done with the stomach drawn up, forming suction through conscious muscular action.

The professional boxers that I used to see in the New York gymnasiums when a boy used to stand interminably, as part of their training, with their hands clasped behind the head, would twist and bend from the hips to the left, forward and down as far as possible without bending the knees, and around to the right in a circular motion, coming back to the original standing position. After some fifty movements from left to right they would do the same exercise from right to left until some two or three hundred movements had been completed. They bent from the hips, mind you, so when they bent forward their stomach muscles dropped. If they were training today and doing the same exercise, instead of bending from the hips they would bend from the diaphragm. They would lock their hips, draw up their stomachs, creating muscular suction, and bend from the line of the lower ribs, never bending back beyond attention position. They would soon develop a new anatomical hinge that would be far more beneficial for their purposes than bending from the hips. In this new exercise the hips are locked tight and the buttock muscles taut. Do it 40 times each way. It can be done in a couple of minutes. Start off with 10 and work up to 40.

Here is another one, the same type of exercise. Draw up your stomach and, extending hands (palms up) as far to each side as possible, make a see-saw board of your shoulders and arms. Reach down and touch side of your right knee with right hand (turning palm in as it goes down). Your left hand, accordingly, reaches up as high as possible (palm still up); but as soon as right hand touches right side of leg it starts up (palm turning up), reaching as high as possible, while left hand starts down (palm turning down) to reach the left side of left knee. This completes the see-saw movement. Each time either hand touches the leg the stomach should be "sucked up" creating the muscular suction that makes the exercises effective and corrective. Don't allow your stomach muscles to drop, ever, while doing these exercises.

Here is number three of the same kind—abdominal. Stand at attention, lock hips, buttock muscles taut. At command "Exercise" raise hands as high as possible, shoulder width apart. Breathe quickly but steadily as arms rise, and after reaching the zenith bring arms down (palms out), directly in front until they touch the knees. Exhalation should start from moment arms start descending and be completed at the moment the hands touch the knees, when the suck-up or

suction action of the abdominal muscles takes place. The action of this exercise resembles that of a jackknife, and the exercise is called the "Jackknife." Should be done 10 times at beginning and worked up to 40.

Here is a fourth, a very simple one, and a very good one. Draw up your stomach as far as possible, take a long step with your left foot, bend over, reaching out with the right hand, fist tightly clinched, until you touch the ground about 8 or 9 inches in front of the toes of the left foot. Don't let your stomach sag. It must be up. Come back to standing position with vigorous jerk, with right arm crooked and muscles taut. The stomach remains up. Do this exercise 15 times, then reverse the procedure by putting the right foot out and having the left hand to do the reaching and jerking back to position. After doing that particular exercise you will find that Spike Webb will want to put you on the boxing team because your hitting power will increase so.

These are are four simple exercises to be done for shortening those stretchy abdominal muscles and strengthening the muscles that control posture.

DEEP BREATHING EXERCISES

Here is what deep breathing exercises have a tendency to do. Watch how erect I stand while I am doing the exercise. That increases lung capacity, brings the stomach up, and makes your head erect on your shoulders.

Deep breathing No. 1.—The exercise is executed from the position of attention. On the first count the student slowly raises his arms, crossing them in front of his body, inhales deeply, throws out his chest, and rises on his toes. When the student's arms are vertically overhead he pauses, and as the second count is given the exercise is reversed. The arms are slowly lowered sidewise of the body completing the arch; heels are lowered to the deck and the breath is exhaled at the position of attention. The procedure is repeated a number of times each morning.

Deep breathing No. 2.—The exercise is executed from the position of attention. On the first count the student slowly raises his extended arms in front of the body, rises on his toes and inhales deeply all the way to the position of "neck rest." The student then lowers his extended arms slowly, keeping the fingers locked, palms out, lowers his heels and returns to the position of attention—unlocking his fingers. The breath must be held until the position of attention is assumed. The procedure is repeated a number of times each morning.

Deep breathing No. 3.—This exercise is executed by filling the lungs to capacity, then at the command "March," 6 paces forward are taken, beginning with the left foot—in this manner: March! Two! Three!

Four! Halt! The stomach must be drawn up, the chest out, the head erect with chin in. At the order "**Halt**" the breath is to be exhaled and then the order "**About Face**" given. After a normal pause the order to inhale is to be again given and after the lung capacity is reached, again at the command "**March**" 6 steps are taken bringing the student back to the original position. This is to be repeated 10 or 12 times each day, 5 or 6 times each way.

There is a final exercise for limbering up. We have found out that doing exercises that cause bending with the knees stiff eventually affects the sacroiliac joint. "**Sacroiliac**" is a very common term. Most people of middle age become acquainted with the term through practical personal experience. It has been discovered that much of the sacroiliac disturbances come from doing bending exercises with the knees stiff. This exercise that I am to do I have been doing for 20 years. I do it for a complete stretch. I hold my legs a foot apart, reach up as far as I can, on my toes, come down rhythmically, and bend my knees, get my hands through my feet and back behind my heels as far as possible, touching the ground there with the back of the hands. Here is the way it is done. The tempo is not rapid but not slow; the exercise is steady even action free from jerks and pauses. It is done 20 to 40 times.

The deep breathing exercises do 5 to 10 times each, and the abdominal muscular exercises do from 50 to 100 times each. This will not take over 15 minutes. And, if you do these every morning before breakfast, Sunday included, I guarantee that you will have no trouble with posture and no trouble with holding your stomach in.

Most other setting-up exercises that we used to do we have found out are not corrective. They don't have the corrective measure that is necessary to shorten those muscles that hold the stomach up.

There is another exercise that officers, and enlisted men for that matter, have access to, or will have very shortly. It is done with an exercising device that I have discovered and given to the Navy. In my opinion the exercise, done faithfully, is the best that I have ever seen. It is not only very beneficial for muscles and muscular tone but it helps heart action and increases lung capacity, which in turn helps posture. Moreover, it shortens that stretchy girdle muscle that is the bane of so much existence. It is a very simple kind of exercise as the demonstration I am about to give will show. The device itself comes complete in a small paper box weighing about 5 pounds, and in a short time can be obtained in Ship's Service Stores. Officers going to sea who have very little opportunity to exercise will find this device a Godsend. It can be put up in the stateroom and every morning used for 15 minutes to get the best results. This will not only meet with the requirements of ALNAV 100 but the device will become so popular and become so much a part of the daily life that one won't be without

it, since it will be discovered to have a very engaging personality itself. If one gets into the cadence of this exercise one becomes attracted to the music of the rhythm. I think that all officers need a little rhythm now and then, particularly at sea.

As you pull your legs up, your hands come down by the side. That looks very simple. It is very simple, but eventually it tires you. The time is, to complete the exercise, 2 seconds. I exhale as I pull my legs up, and inhale as I let them down. Now you can do that 500 times in less than 15 minutes. That is the exercise that we guarantee will keep a man in shape and develop the proper posture. Also guarantee to take 2 inches of the waistline in 60 days. By the end of 6 months it ought to take 6 inches off, if I know my arithmetic. That, however, is not part of the guarantee. It really is about the finest type of exercise for all types of people yet invented.

The Secretary of the Navy is very serious about this ALNAV 100. We want an aggressive, hard-hitting organization of fighting men, and that is the purpose of this ALNAV 100 and the physical fitness program. We want to be ready for whatever happens. We find, the Navy finds, and I suppose everybody else believes, that in order for a man to give his best and maximum efficiency he must be physically fit to begin with. To make all personnel of the Navy, officers and men alike, physically fit, should be one determined purpose.

UNITED STATES NAVAL MOBILE BASE HOSPITAL NO. 1

By Captain Lucius W. Johnson, Medical Corps, United States Navy

This hospital was established by order of the Secretary of the Navy, dated September 9, 1940. The material and personnel were embarked on a Navy transport October 25 and reached its base in the Caribbean area October 30. We were subsisted on the transport until after breakfast, November 2, when our own mess was established.

The hospital had a definite task as a part of the mission of the Medical Department. This task was:

- (a) To erect the hospital;
- (b) To provide the best possible care of patients;
- (c) To keep the hospital as mobile as possible, so that it could move quickly;
- (d) To inform the Bureau of Medicine and Surgery concerning the suitability of our equipment, personnel, organization, and administration.

PRELIMINARY WORK

During the time that our material was being assembled on the dock at New York, the carpenter's gang was employed in building

tent floors, sorting out the parts for the prefabricated buildings, and marking them so that the parts for each building could be quickly segregated. Blueprints of the many machines were studied, and plans prepared for setting up the various apparatus.

A plan of stowage was worked out, so that the articles which would be urgently needed for immediate use on landing should be the first ones unloaded. These careful plans for last-in, first-out stowage went so far awry that it would have been ludicrous if it had not been so serious. The reasons for this are worth telling, so that others may avoid them, though most of them were not within our control.

The tents, tools, ranges, latrine boxes and other articles which would be immediately needed were placed, by arrangement with the ship's officers, in the top layer of No. 6 hold. We saw the hatch closed and left for the night, happy in our feeling of intelligent planning. But the stevedores worked long hours that night, stowing piece lumber on top of No. 6 hatch. On arrival at our base, the word was passed to unload heavy articles first, so that the dock could be kept clear. Nothing moves more slowly than piece lumber, so work was not started on No. 6 hold until late in the day. Instead of our articles most urgently needed, the first to be unloaded were caskets, ward furniture and other unwanted objects.

The limited docking facilities further complicated our situation. It was necessary to clear the dock for another ship which was to arrive shortly after the transport. The Naval Station and the Fleet Marine Force pressed all their trucks into service to move our goods. They were brought to the hospital site, about 2 miles away, too rapidly for our people to identify and segregate them. Thousands of boxes, bales, barrels, cartons and crates were dumped promiscuously about the 8-acre site. This work was carried on until midnight the first day. For a time, a motor-driven floodlight was provided, but this soon ran out of gas, and for the greater part of the evening, the work was done in darkness, so that no intelligent supervision of the unloading, or identification of the articles was possible. This indiscriminate dumping meant that many days of labor by a large gang were necessary to find the articles that were urgently needed in order to establish ourselves. It also meant that thousands of pieces had to be worked over and handled repeatedly in the process of sorting. Many of the most-needed items were not found until well into the second week. During the first few days, we had too few shovels, picks, saws, hammers and other tools for digging, leveling, clearing, opening boxes, driving tent pegs, making tent floors and many other necessary jobs.

Our trucks were badly needed to help move our property to the hospital site, but the 5-ton and two of the 3-ton trucks were unloaded to a lighter on the outboard side of the ship. No tug was available



1. ENLISTED STAFF QUARTERS. IN TENTS.
2. RECREATION AREA; MOVIE SHACK AND SCREEN; VOLLEY-BALL COURT.
3. TENT WARDS; 25 PATIENTS IN EACH WARD; UTILITY TENTS AT ENDS OF WARDS
4. MEDICAL WARD, IN PREFABRICATED BUILDING.
5. OPERATING ROOM AT LEFT. AT RIGHT, SURGICAL WARD IN PREFABRICATED BUILDING.
6. TENT SURGICAL WARD, OPERATING ROOM AT RIGHT.
7. BUILDING CONTAINING DENTAL CLINIC, LABORATORY, PHARMACY, AND COLD STORAGE.
8. FIVE MESS HALLS IN PREFABRICATED BUILDINGS; GALLEY AND WATER HEATERS BETWEEN BUILDINGS; BAKE OVENS AT RIGHT.
9. X-RAY BUILDING, WITH GENERATOR SHACK AT RIGHT.
10. TWO 20-K. W. GENERATORS.
11. MAINTENANCE AREA. ELECTRICAL, MACHINE, AND CARPENTER SHOPS, LUMBER PILE.
12. ADMINISTRATIVE AREA. OFFICES ON EACH SIDE OF CENTRAL FLAGPOLE.
13. STORAGE AND ISSUE TENTS; BAGROOM; BULK STORES UNDER TARPULINS.
14. INCINERATOR, HILLSIDE TYPE.
15. LAUNDRY.
16. OFFICERS' QUARTERS.
17. WATER-PURIFICATION PLANT AND STORAGE TANKS



PYRAMID TENTS AS QUARTERS FOR THE MEN. NOTE STRONG BACKS FOR SECURING CANVAS.



UNLOADING FROM TRANSPORT. CONGESTION ON DOCK.

to move the lighter to the dock, so we lost the use of the trucks until nearly 1,700 that day.

These and other incidents are recited, not with any thought of complaint or criticism, but with the purpose of showing how unforeseen complications and difficulties can interfere with the best-laid plans. Precautions can be taken against those that happened to us, but other problems will arise to vex our successors in this work, so allowance should be made for them.

Looking back at our experience with the Mobile Base Hospital, one of the things that stands out most prominently is the number of grand ideas that went wrong. They looked foolproof and wonderful as we talked them over, but when we tried them out they turned sour and didn't work at all. Some unforeseen condition always turned up to interfere with the smooth working of our enterprises. Eventually, we adopted as the slogan, "How could we be so dumb?"

THE HOSPITAL SITE

This was on a point extending about 1,000 feet northward into a bay. Its greatest width was about 350 feet at its southern border, from which it tapered irregularly to a rounded point. The base was coral, over which there had been a lava flow of varying thickness. Above this was a topsoil with extreme depth of 18 inches, and many outcroppings of rock. The area was originally covered with a dense growth of cactus and thorn bushes. Before our arrival it had been cleared by using a bulldozer and pushing all the chaparral over the edge of the cliff. This great mass of dried brush harbored countless mosquitoes and other insects, and was also a fire hazard. One fire in this brush gained such headway that we were compelled to call on the Naval Station for help with their fire apparatus. A gang of men and a truck worked for 3 months to clear the brush from the borders of the hospital site.

The trade winds blew strongly across the area by day and by night, so that everything was enveloped in a cloud of dust. This was corrected, to some extent, by hauling several hundred truck loads of gravel from the beach, 3 miles away, and spreading it over the roads and vacant spaces. This added materially to the comfort of both patients and staff.

The space officially assigned us contained about 20 acres of fairly level, useful land, and we laid out the hospital on that basis. On visiting it, however, it was found that two deep gullies traversed the site; a main road had been cut through it; permanent buildings had been erected on it, and the shore lines shown on the map were inaccurate, so that the useful area was reduced to about 8 acres. This was much too small for our needs. Even with close crowding there was

not room enough to set up 500 beds in tents, with our other facilities. There was not space enough for the wide separation of hospital units, which should be the rule if we are to take advantage of the lessons learned abroad, as a result of air attacks on hospitals. This experience suggests that one of the first steps in establishing future mobile base hospitals should be to make an accurate survey of the site, and lay out all wards and other structures, also roads, water, sewer, and electric systems.

PROGRESS NOTES

1940

October 30.—Arrived at our base. Unloading commenced.

November 2.—First meal at our own mess, 1200. First night in tents. Water line completed to our property. First chemical-tank toilet ready for use. Fifty-one tents erected.

November 3.—Last of our freight delivered to hospital site at 2100. Showers for men in use. Four heads ready for use. Electric lights for mess tents and officers' quarters.

November 5.—Water-purification plant completed and in use. Public Works Department of Naval Station commenced work on laundry. First water-tank tower completed.

November 6.—First water tank filled with treated water. First portable building footings complete and floor laid. Recreation tent provided for men.

November 7.—Second water-tank tower completed and erected. First ward ready.

November 9.—Public Works poured concrete wall of laundry. First prefabricated building ready for use as galley. Beds installed in first ward.

November 13.—Second prefabricated building completed and in use as mess hall. Seventeen pyramidal tents complete with floors for crew. C. P. O. showers and toilets in use. First day of operation as hospital, 5 patients admitted. First-aid and sick-call tent, also laboratory and pharmacy tent in use.

November 14.—Additional material arrived. Second mess hall completed and in use. Equipment moved into x-ray building. Eight office tents completed.

November 16.—Public Works completed pouring floor of laundry. Thirty-eight pyramidal tents complete with floors for men. Floors for office tents completed.

November 20.—First operating-room building completed. Concrete foundations for galley boilers completed.

November 25.—Surgical-ward building ready. Fifty pyramidal tents with floors now complete, for crew's quarters.

November 29.—Dishwashers installed. Excavation for cold-storage



TENT WARD.



PREFABRICATED BUILDING BEING ASSEMBLED.

plant complete after five days work by two gangs with air hammers. Tent wards 3 and 4 complete.

December 2.—Poured concrete for cold-storage building and garage slab.

December 4.—Visited by Surgeon General.

December 6.—Dishwashing plant complete. Screened galley complete. Eleven wards now ready for use.

December 10.—Visited by Secretary of the Navy.

December 11.—Poured floor of cold-storage building, also concrete floor for screened galley. Three tents for maintenance supplies completed and occupied.

December 13.—Laboratory and pharmacy ready for use. Sinks will have to be made by hand. Five Homelite generators burned out so far.

December 16.—Four wards in use, with 75 patients.

December 19.—Inspection by Commander Train, Patrol Force, and staff.

December 20.—Laundry building complete. Public Works men still at work assembling machinery.

December 23.—Cold-storage building completed. Machinery being installed.

December 26.—Men's recreation tent completed. First moving picture shown.

December 31.—No. 3 mess hall completed. Laundry machinery installation 40% complete in 6 weeks of work. No. 2 operating room completed.

1941

January 2.—Portable building for medical ward completed and occupied.

January 6.—Installed 20-kilowatt x-ray generator on its base.

January 10.—Installation of cold-storage boxes complete, and being tested.

At this time there remained only the completion of the cold-storage equipment, and the laundry. There was a smile on every face the day that ice cream from our own plant first appeared on the menu. It was interesting to observe the universal popularity of this food, which even surpasses pie as a dietary favorite. The laundry was not completed until 3½ months after our arrival.

By the middle of January we had 171 tents complete, 50 of them in use as quarters for the enlisted staff, 75 for wards, 10 for offices, 13 for storage, and 22 for officers' quarters. There were also 12 prefabricated buildings, 5 used as mess halls, one for the laundry, one for dental clinic, laboratory, pharmacy, brig, and cold storage, also 2 for operating rooms. Our number of patients rose to 140.

PERSONNEL

One of the important lessons that we have learned from our experience in setting up the first mobile hospital is, that it is not good economy to have the labor of construction performed by men of the Medical Department. Our men have done the work of stevedores, ditch diggers, cement workers, pipe fitters, carpenters, and laborers. No word of protest has been heard, no matter how hard the work, or how long the hours. The traditional versatility of the hospital corpsman has been demonstrated.

For 2 months our chief of the medical service has supervised the erection of tents. Our urologist has been in charge of stores in the dump. Our laboratory officer has been in charge of laying water pipes, and concrete work. Our x-ray specialist has been in charge of digging latrines. Our psychiatrist has become expert in planning and erecting shower baths. All sorts of latent talent have been developed, but in men whose relatively large salaries are justified because of their special training in medicine. It is a matter of general knowledge that there is a shortage of these trained men in the Navy.

We believe that, when another mobile hospital is planned, it would be much better to provide artificers and laborers to do the work of construction, under the direction of the engineer and one of the senior officers of this hospital. The medical stores, medical staff and hospital corpsmen should be sent about the time the buildings are ready to be occupied. In the meantime, their services would be available in some naval activity. This was the plan followed in construction of the United States Naval Base Hospital at Queenstown, Ireland, during the last war. There has been opportunity to contrast this method with that of having the hospital corpsmen do the labor. The decision must be in favor of the former.

Personalities.—There is a certain type of individual who is frequently out of favor with his fellows in the Navy. He may be at odds with the authorities, or merely regarded with some uncertainty by his associates. In a large hospital such men may be wasted or unwanted, because the administration is so far removed from the individual that nobody has time to study them and find out why they differ from the others.

In a pioneer outfit like this, such odd numbers may prove especially valuable. They are often different from the pattern because they have more energy, more imagination, or less self-control. Their energy and imagination are at a premium when such a great amount of physical labor is to be accomplished, and they often turn out to be excellent leaders. A certain proportion of problem children can well be utilized in organizations like this. These remarks apply alike to officers and men.

Disbursing officer.—The necessity for an officer of the Supply Corps would never have been appreciated if we had not been for 3 months without one. Our difficulties with pay, money orders, clothing, and small stores, procurement of provisions and other details leave no doubt that such an officer should be a part of the personnel from the very beginning. Some of the many problems of supply and disbursing for this hospital arose from its amphibious nature. While it was a part of the Atlantic Fleet, it was located temporarily ashore, within a naval station to which it did not belong. An officer of the Supply Corps eventually joined us as supply and disbursing officer. His article, in this issue of the *BULLETIN*, tells of some of the difficulties that he encountered, and the manner in which they were adjusted.

Civil engineer.—He proved invaluable in laying out the structures, superintending construction, and general planning. We were fortunate in having a young Reserve officer with wide experience in all sorts of construction work, and a very practical turn of mind. The civil engineer should be one of the first officers ordered for duty with future mobile hospitals. His important duties of laying out the buildings, roads and other facilities provide the fundamentals for the development of the whole institution. This officer has also contributed a short account of his experiences which is published in this issue.

Non-hospital ratings.—Their services were of great value, though their number was far from sufficient. It is suggested that, when a new mobile hospital is planned, outside the United States, it might be possible to borrow enough artificers from other naval activities to do the construction work, and return them after it is finished. My experience with the hospital at Queenstown during the last war proved that construction by laborers brought up in foreign ways of doing things was most uneconomical. It would be much better done by Navy men under Navy supervision.

The complement of artificers was far below our needs and this resulted in failure to complete vital units during the first 2 months of construction. Large number of Hospital Corps men worked in the construction gangs. As the wards were opened and our patients were increased, it was necessary to take the corpsmen off construction work and so it progressed more and more slowly. Our original allowance of non-hospital ratings was just about enough to provide for maintenance after the construction was completed.

Hospital Corps.—Our complement was inadequate because so many duties which are performed in other naval hospitals by female nurses and civilian employees were here done by Hospital Corps men. Tent wards required constant cleaning and sweeping because of dust or mud. Excretions of ward patients had to be carried a long distance to the deep-hole privy. Food had to be brought from the galley, a long

distance, and the dishes and waste returned there, all by the Hospital Corps men.

Other duties of Corps men, not usual in naval hospitals, were: Procuring provisions, which included sending working parties to ships to break out stores; incinerator detail; procurement of fuel, delivery to hospital and distribution to many power units; operation of laundry; sanitation, clearing of brush, mosquito control; operation and maintenance of motor transportation; mess cooking and guard duty. In addition, it was necessary to supplement the inadequate number of non-hospital ratings by detailing Hospital Corps men to construction work.

Dental Corps.—The one dental officer detailed to this unit proved to be sufficient, though additional ones would be required in case of combat, to care for injuries in their special field. Our dental officer performed many general duties, far outside the scope of his profession, in a most competent manner.

Medical Corps.—The number provided was adequate to care for the number of patients that we had. The selection of individuals seemed ideal, for there was not a misfit in the lot. The commanding officer often had the feeling that, if he could have hand-picked every officer and man in the organization, he could not have gathered a finer group than came from random selection of those available within the Medical Department. For every problem that arose, some member of the group was found, who was equipped by experience to show us the solution.

General.—It is important that only young and vigorous men be sent with a mobile hospital. Many fleet reservists who have been out for several years are too old for this work. As an example, five of our men may be cited. They were elderly men of fine character and great ability in their lines. They were determined that none of the young men should do any more work than they did. They were too proud to shirk or to admit that they were tired. As a result, they broke down and were seriously sick for a long time. Fleet reservists called back to active service should be assigned to duties that are suited to their strength.

HOUSING

This was spoken of as a 500-bed hospital, but the housing provided for only about 350 patients, and the site was not large enough to accommodate many more than that number. We had both tents and portable houses, and had plenty of opportunity to observe their suitability for our uses.

Military men agree that tentage is the most expensive means of housing troops. Its initial cost is high, and the average life of canvas when exposed to the weather is 6 months, after which there is nothing

worth salvaging except the poles. Our tents began to show extensive mold discoloration after about 4 months.

The advantages of tents are that they are easily transported and quickly set up, so that they add materially to the mobility of a unit. For use as wards they have very definite disadvantages. They are hot and dusty in the day time, cold and damp at night. The fire hazard is great. The flapping of the tent is annoying to the patients and interferes with physical examinations. It was necessary, because of the numerous insects and the prevalence of malaria, to use mosquito nets on all beds. The nets added greatly to the difficulty of nursing care, and they increased the discomfort of sick patients. Many pieces of ward furniture, such as cabinets, ward screens, refrigerators, irrigator stands, and linen locker, are so tall that they can be placed only in the midline of the tent. Several of our patients, admitted for other causes, developed pneumonia while in tent wards.

For wards we used six 14- by 14-foot wall tents, placed end to end. We found it necessary to use one of the end tents as an office and the other as a utility room. Later, we set up a utility and mess tent for each 2 wards. This worked out very well and gave us an additional 5 beds in each tent ward. In each tent we placed 3 beds athwartships on one side, and 2 beds lengthwise on the other. This gave us 5 beds in each tent, and also threw the aisle off center, so that the poles were out of the way. We believed that a 7-tent ward, providing an office and 30 beds, would have been a better arrangement, but our site did not lend itself to this arrangement.

Two tent wards were placed side by side, with an intervening space of 5 feet. The pairs of tent wards were separated by a street which was 8 feet in the clear, giving plenty of room for the passage of a fire cart. The pegs must be placed 5 feet from the edge of the tent, otherwise the side of the tent will be so low that a bed, or ward furniture, cannot be placed close to the wall, and the useful area would be greatly reduced. If strongbacks are used, the tents can be placed much nearer together, but this interferes with the ventilation and the comfort of the patients.

Complete data were compiled for constructing and erecting various types of tents, tent floors, strongbacks, prefabricated buildings and framed, screened structures. It is not considered suitable to include them in this report, but they are available for the guidance of those planning future mobile hospitals.

Wards in prefabricated buildings have many advantages. The walls are high enough so that hospital beds of the standard height can be used. They are screened, so that individual nets are not needed. Nursing care and physical examinations are much easier. The side walls protect against the clouds of dust that sweep through the tents. The hinged shades protect from the direct sun, but allow

full ventilation. The comfort and well-being of patients are much better in buildings than in tents.

These buildings are described by the manufacturer as portable, but the term "prefabricated" gives a more accurate mental impression. They are portable until they are set up, but once they are erected and the plumbing and other fixtures attached they can not be taken down and shipped to a new site without serious damage.

Prefabricated buildings are also superior to tents for quarters, storage, mess halls, galley and offices. Unless a hospital is to have the complete mobility of a field hospital, prefabricated buildings should replace tents throughout. The original cost of the various types of housing per square foot of floor area was approximately \$0.50 for tents, \$1.50 for the screened type of prefabricated building, and \$2.90 for the building with interior lining of walls and ceilings.

COMMISSARY DEPARTMENT

To provide suitable food for staff and patients in the field is as difficult as it is important. One must get along without many of the facilities which help so much to make the work easier in fixed hospitals. For 72 hours after arrival here, we were subsisted by the transport. Though not completely satisfactory, this was a great advantage. Our early meals ashore were prepared and eaten amidst a cloud of dust and flies. Sixteen days after arriving, our first screened mess hall was completed and occupied. From the twenty-third day (Thanksgiving Day) on, all hands were fed in screened mess halls. On the thirty-first day the screened galley was complete, and the flies were no longer a serious problem. We were fortunate to escape any serious outbreak of gastrointestinal disease during the early days.

At first, each man was issued a set of Army field mess gear. They were served cafeteria style, and after the meal each man washed his mess gear in G. I. cans of boiling water. This was a simple method and required the minimum number of mess cooks. On the forty-fourth day, the dishwashing machines were put in commission, and the men were served at tables, using Navy mess gear.

Since food is such an important item in maintaining morale, we were exceedingly fortunate in our commissary personnel. Our commissary officer had just completed a course of instruction in that work. Our chief commissary steward had been for several years in charge of the restaurant department of one of our largest chain stores. Together with their assistants, they never failed to turn out the finest of food, with plenty of pie, cake and other desserts. They were handicapped by poor stoves with unsatisfactory fuel, inadequate refrigeration, dust,

flies, and general lack of facilities and conveniences but, from the very first meal, their food was excellent.

All officers and men were subsisted in the general mess, and we found it so satisfactory that no extras were needed.

WATER

In the course of a preliminary visit to our base, I was assured that water would be brought to the site and an outlet provided before the hospital arrived. We found, however, that this had not been done. It was necessary for us to purchase pipe and fittings at a cost of about \$400, and for our staff to lay 1,700 feet of 2½-inch and 2-inch pipe to bring the water to our site. This took 4 days.

For water treatment, we had a Wallace and Tiernan four-tank machine. The water was treated with chlorine, alum and soda ash, filtered through four sand and gravel tanks, and pumped to storage tanks. A gasoline engine was the motive power, with an electric motor as stand-by. It took about 4 hours to pump the 9,000 gallons which was our average daily consumption.

This purification unit was assembled and we saw it running at the plant before it was packed for shipment. As a result of this inspection while actually operating, the plant was complete in every detail. We had no difficulty in assembling it, and putting it to work. If this same inspection of the laundry machinery, the cold-storage plant, the heaters for the dish washers, and the x-ray equipment had been made, it would have demonstrated that many essential parts were missing, and others were not suited to the requirements. I believe that such an inspection under actual operation should be made of all machinery for future mobile hospitals.

ELECTRIC POWER

It has been strongly impressed on us that an adequate power plant is a basic need of a mobile base hospital. Electric power has many advantages over others. The units are compact and can be mounted on skids or wheels so that they are mobile. Fuel is available for them from any ship or station. They are safe to operate, and almost fool-proof. They can be concentrated in one place, so that the number of watch standers is the minimum. One or more units can be cut in or out to provide the desired amount of power.

At first we had stoves and ovens operated by coal and wood; lanterns, sterilizers and generators operated by gasoline; lanterns and refrigerators burning kerosene; motor generators using Diesel oil; steam boilers burning fuel oil. Procurement, storage and distribution of these various fuels required much time and labor. If all could be

operated by electric power from a central power unit using a single fuel, it would make a great saving.

All refrigerators should be electrically operated. Electric stoves are now so well developed that they are quick, clean and economical. Their bulk and weight are less than stoves using other fuel. Gasoline sterilizers are a serious fire hazard in tents or wooden buildings, as are kerosene refrigerators. We had more than one narrow escape from such a fire. Both should be electrically operated.

I recommend that, when the next mobile hospital is planned, all possible lights, machines and facilities be operated by electricity. After all these are selected and located on the plan, the complete data and requirements should be submitted to some bureau that is competent in such matters, with the request that they recommend generators of the size, number and type that will best fill the requirements.

MOBILITY

It should be borne in mind that this hospital was an experimental unit. It was our task to find out what was the best equipment, the best means of transportation, the most suitable personnel, the most appropriate methods of organization and administration. Extensive recommendations on these subjects have already been made to the Bureau of Medicine and Surgery. Other bases now being assembled will be infinitely better because of the errors and faults of No. 1.

"Mobile" is a relative term, not a precise one. In a hospital designed to serve the armed forces, mobility may vary from that of the field hospital, which should be ready for patients in the minimum of time after landing, to that of the base hospital, which is usually fixed for the duration of a campaign. The present hospital was mobile to the extent that it was transported from New York and was ready to receive a limited group of patients on the 15th day after arrival at its new location.

Mobility is determined by the equipment. A field hospital has only meager equipment and is designed to render only a very limited degree of treatment, hence it is very mobile. A base hospital is designed to provide a much more elaborate service, and so it is firmly anchored by the weight of its equipment.

A hospital should be exactly as mobile as the military force that it is designed to serve. The degree of mobility that is desired should be one of the first decisions made, and then the equipment should be selected on that basis. Ability to move in two hours, or 2 months, or any other time, can be achieved in this way.

For a number of years the Marine Corps and the Medical Department of the Army have been engaged in active research, seeking portable or automotive units which would increase the speed of movement

of military forces. The Navy has improved its means of loading heavy machinery and stowing it on board ship. At an early stage in choosing equipment for this hospital, it was believed that the Army's mobile units were too large and heavy to be moved by Navy ships. I have since investigated this point and find that certain Navy store ships can load and stow units up to 30 tons in weight, which is much heavier than any of the Army's tractor-trailer Medical Department units. The largest tanks and tractors are handled without difficulty. This would make it possible to employ a number of the Army's tractor-trailer units and carry them in the forward hold of such a ship.

Acclimatization of the men is also a factor in mobility. When our men left New York, they were pasty-faced and flabby from prolonged city life. Immediately on arriving, they were called upon to perform the hardest kind of labor for long hours in excessive heat. They perspired profusely and drank great quantities of water. They were required to protect themselves from severe sunburn. Countless flies swarmed about them and the trade winds stirred up a continuous cloud of dust. About the third day men began to fall out from exhaustion and severe diarrhea, but a month later they were bronzed, stalwart men, in the most enviable state of health.

CLINICAL WORK

In addition to the experimental work on equipment and facilities, care of patients was an important feature. Our patient load increased until, in February and March 1941, it was stabilized for some time around 240 patients. This required an organization somewhat different from that of the usual naval hospital in a fixed location. There are many difficulties in clinical work in a tent hospital in the field, which one would not appreciate until he had tried it. These were attacked with enthusiasm by our staff, and satisfactory ways of carrying on were developed. With more than 1,000 patients admitted, there were only 2 deaths, both from malaria complicated by pneumonia.

The interesting fact was established that, for Marines in the field who are not engaged in actual combat, hospital beds will be required for 3 percent of the force. This number would vary, of course, with climatic conditions, and with more seasoned men. Two-thirds of the men at this station were described as fresh-caught.

Soon after our arrival, cases of malignant tertian malaria began to appear. Half a mile to windward of our site we found several hundred native laborers housed without screens. Between the hospital and their barracks there were at least 20 pools of stagnant water, teeming with anopheline larvae. The medical officers of the 3 military units which were exposed to this danger of infection immediately combined to control this public health hazard. The necessary steps

to prevent mosquito breeding were inaugurated, and a malarial survey of the native laborers was arranged. In our laboratory more than 3,000 blood films were examined. The whole episode provided a very pleasing illustration of the ability of naval medicos belonging to different military organizations to cooperate smoothly in the face of a common danger.

THE "MOBILETTE"

While our hospital, as a whole, proved to be less mobile than we had hoped, we were able to send out a completely mobile unit of 100 beds to accompany a force of Marines which went for several weeks of maneuvers to a base several hundred miles away. This unit, with its complement of medical officers and men, established its hospital in a suitable location and operated throughout the exercises. In addition to the actual needs for hospitalization of patients, they practiced constantly with first aid and transportation of constructive casualties. An epidemic of a mild contagious disease on one of the ships filled their beds almost to capacity and provided valuable information about the needs of such a mobile unit.

There are several prospective areas of combat where the combination of a relatively fixed base hospital, with highly mobile units which it can send out to serve advanced bases would be most useful. Evacuation to the base hospital could be by air or by ship. Alaska, the Pacific islands, and the Caribbean islands are examples of such areas. The mobile base hospital offers a satisfactory solution at a relatively small cost.

MOBILE BASE HOSPITAL NO. 1

LOOKING IN FROM THE OUTSIDE

By Commander Thomas B. Magath, Medical Corps, United States Naval Reserve¹

On the assumption that an outsider's viewpoint is sometimes interesting and occasionally helpful, I am recording my impressions of the interesting and useful experiment in the erection of Mobile Base Hospital No. 1. I had the pleasure of landing there one warm sunny afternoon in the middle of January after a large share of the construction was finished and since my active duty in the Navy had been minimal and my indoctrination less, these observations may be considered strictly objective, unorthodox, and probably different from those of the regular naval medical officer.

It did not take long to realize that an enormous amount of labor had been expended by the whole staff, men and officers alike, in the construction of the hospital. While I was not there to hear what might have been said during the hot waterless days, the mosquitoey nights,

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and the plague of flies, it was evident that the hardships had not damaged the morale of the group and the loyalty to the organization was keen. The ability to joke about the numerous inconveniences had been developed to a high degree, indicating a healthy mental state.

Of the hospital itself I was struck by the completeness of the service. With the exception of certain types of mental cases almost any medical care could be rendered and well rendered. The laboratories especially pleased me, for any necessary diagnostic test could be executed and adequate control of treatment was available from a laboratory standpoint. Attempts to prepare solutions to be administered intravenously did not meet with success and it is evident that under these conditions commercial products, now excellent, should be provided. In regard to roentgen ray equipment it would seem that if a hospital is to function as a base, thereby implying sustained action, screening should be provided for the protection of the technicians. The pictures made with the apparatus, however, were excellent and certainly no clinician could complain that these two services were not adequate for the hospital.

The surgical units, housed in portable, lightly constructed buildings, while functioning excellently, furnished an admirable example of the necessity of adequate control of sterilization. While this was covered to some extent by placing soil in the sterilizers and testing it after sterilization, the method is laborious and is not practical for ordinary work. It would have been far better to have used one of the excellent ink control tags, so that each batch sterilized would have been known to be sterile. The method of heating the autoclave with kerosene burners was slow and in subsequent installations either one of the tanked gases or electricity should be considered for the source of heat. It might be possible to work out a scheme by which the steam pressure boilers used for sterilizing dishes could be used to operate the sterilizers. It was also quickly recognized that double screen doors should be provided for the operating room in order to eliminate the possibility of flies entering the room when the door was opened.

The medical specialties and dentistry from my viewpoint were adequately housed, being for the most part in tents, and if one accepts this housing as correct for a hospital of this type no further comments are necessary. It should, however, be pointed out that in semitropical and tropical countries the problem of mosquito transmission of yellow fever, malaria, and filariasis is of considerable concern. In tent housing it is necessary to screen each bed separately. Even then patients sleep against the screens and may be bitten through the screens by these transmitting agents; hence individual screening of beds cannot be looked on as absolute protection against these diseases.

It is probably unnecessary to call attention to the situation in regard to the ambulances. They were magnificent looking modern city ambulances which seemed a bit incongruous when a patient was unloaded from one onto a cot in a tent after a run over a muddy road. However, this view was so generally accepted by all hands that an outsider did not need even to refer to the matter. The quarters for the officers and the men, galley, mess rooms, and various mechanical services certainly were adequate and in general everyone seemed to be comfortably housed and very well fed. One cannot help but call attention to the excellent diet which was provided for the officers and men and the satisfactory way in which the food was handled and prepared. It was hard to believe that the men involved were not hand-picked for the occasion but an examination of the record did not indicate this to be the case.

HYGIENIC PROBLEMS

In all projects of this nature the sanitation of the camp will be of primary importance and this will depend to a great extent on the general condition of the community. There were a number of interesting problems at the Bay which had a direct bearing on the sanitation of the base hospital. There was, for example, the question of water supply. Water was obtained from an obviously contaminated source and was treated at various points near its ultimate distribution. The ultimate supply from a standpoint of sanitation was certainly adequate as to both quantity and quality. The problem of the vulnerability of the distribution system, however, was by no means solved.

Since there was no general sewage disposal plant, each command provided its own method and this varied widely throughout the area. The disposal of sewage embraced cesspools, various types of latrines, dumpage into the bay and other less satisfactory arrangements.

The garbage disposal problems presented all of those found in a city of comparable size, and no solution had been provided that could be considered adequate. A large dump infested with rats and flies was maintained near the water's edge and some garbage was disposed of by dumpage at other points in the area. A small amount was fed to hogs but the arrangement was not satisfactory.

A specific problem which appeared after the Mobile Base Hospital was erected was that of the control of malaria. This area, having not been more than sparsely populated in the past, apparently had been free of malaria for an indefinite period. With the introduction of the large numbers of nonimmune persons and with the bringing over of a large number of workers from nearby Cuban villages, both benign and malignant tertian malaria began to appear. Under such

conditions, with adequate breeding places for anopheline mosquitos, a fair-sized epidemic of malaria was easily predictable. One of the serious aspects of such an epidemic would be the transmission of the parasites back into the United States, and into areas which previously had been free of malaria, because of the shuttling of troops. The possibility of introduction of other strains into the area by the movement of troops through the islands was also an important problem. Up to the present time the only mosquito which is known to be of importance in the transmission of malaria in this area is *Anopheles albimanus*. This mosquito breeds for the most part in water which is unshaded and, hence, the usual procedure of cleaning out brush to stop mosquito pests is unsuitable for control of malaria in this area. If this site is to be kept as a permanent base with a fairly large population, the only satisfactory control of the malaria problem is permanent drainage, using the tile construction already successfully demonstrated in Cuba, and the planting of shade trees along the streams. Fortunately the terrain is of such a nature that this could be cheaply done and ought to reduce the hazard of malaria to a minimum in this region.

The problem of mental hygiene is a real one at every station and everyone recognizes that the provision of an adequate recreational program is not only desirable but of prime necessity. The lack of such a program was a distinct handicap.

HOW THESE PROBLEMS WERE MET

When these problems were specifically applied to the Mobile Base Hospital the solution had to be a partial one.

The water supply for the hospital, which consisted of a portable treatment set, functioned excellently. The only criticism seemed to be that there should have been an additional chlorination of the water after it had passed the filters, but the criticism is certainly not serious and many sanitary engineers consider one prechlorination quite sufficient.

In regard to the sewage disposal, chemical toilets were provided which were satisfactory from a strictly bacteriologic standpoint, in that the effluent, being so highly alkalized, was sterile. This method, of course, would be limited to a small group and the expense of maintenance would be prohibitive if extended over a long period. The question of disposing of sewage into the bay was considered by many officers. Fundamentally this method would never be approved by sanitary engineers and yet on this specific occasion it did seem to be justified, provided the effluent was carried far enough away from the shore and provided swimming was strictly forbidden. Anyone could see the inconsistency of having a large number of ships discharge their

sewage into the bay for long periods and then prohibiting the station from using the same method. Nevertheless it does seem justifiable to consider the possibility of submitting the sewage to at least primary treatment before discharging it into the bay and this should be a matter for future study. The small tide and the natural protection of the bay against wind make the problem a difficult one.

The garbage disposal, although a bit laborious at the hospital, was certainly as satisfactory as any in the area. It consisted of burning the garbage in the open. This prevented a rat nuisance and a fly nuisance as well, but was difficult to carry out in rainstorms. If the hospital is to be permanent or semipermanent, some form of cheap incinerator could be used to advantage, although a far better solution would be an incinerator for the entire area.

Careful study of the areas immediately surrounding the hospital did not reveal breeding places for mosquitoes except during the first days when the hospital was set up and these areas were quickly eliminated. The use of bed screens for all officers and men, of course, reduced the hazard tremendously, but the showers and toilets were not screened and these furnished potential sources of infection, especially in the evening. The screening of the mess halls and galleys was effective.

The fly nuisance around the hospital was well taken care of by extreme care in regard to general cleanliness. In subsequent construction of hospitals of this type, however, a screened passageway between the galley and the mess hall should be provided as additional protection. The care of utensils and dishes was excellent and would have been envied by some of the best hospitals in this country.

The recreational program of necessity lagged behind the other activities of the hospital because of the urgency of finishing the construction. Obviously the program of any one unit in the area should be correlated with the general recreational program of the whole area and the solution of the general problem will undoubtedly be one of the most difficult at the station.

I was particularly interested in the informal meetings which were held in Captain Johnson's tent on any and every occasion. It was understood that the staff was welcome to come to the commanding officer's personal tent for conferences at almost any time and he was more than receptive to suggestions and host to many groups. The necessity of numerous conferences among hospital staffs has been recognized by all and it occurred to me that these informal conferences were far more valuable than a more formalized type of staff meetings, although these also have their place. To a reserve officer one cannot overemphasize the value to be gained from these informal discussions with men who have been long in the service and whose experience has taught them many valuable lessons. The experience in this par-

icular hospital furnished probably the best opportunity for training of reserve officers that could be obtained, since it certainly was a hospital in the raw and all procurement, construction, organization, general and specialized services, and correlation with other groups in the area, gave one the widest type of opportunity to observe the solution of these problems in the shortest possible time. I cannot refrain from calling attention in this connection to the helpful suggestions and kindly instruction given by all the officer personnel of the hospital. The executive officer, Commander J. H. Chambers, was particularly careful to make many details clear and the wide experience and excellent professional knowledge of the others made a lasting impression on me.

FURTHER PROBLEMS

For a novice in military affairs to discuss the more general aspects of a mobile base hospital would be the height of presumption and yet there were questions which constantly occurred to me in the development of this unit. Certainly the function and purpose of the unit should be clearly defined and it is assumed that out of the experiment a clearer definition has been proposed. If the unit is to operate as an advance base its differences in function from the field marine hospital ought to be definitely set forth. The mixture of portable and nonportable pieces of apparatus and buildings made one doubtful as to the purpose of the unit in a theater of war. Obviously the laundry, which consisted of many heavy pieces of equipment placed on concrete floors—pieces which after several months were still not installed—could hardly be considered a part of a mobile unit. If large refrigeration units are to be provided which are to be run by electric power it would seem reasonable to use electricity for baking and cooking. On the other hand, if the hospital is to be quite mobile, then portable electric units on wheels should be provided which are not to be placed on solid concrete piers and the laundry probably will have to be dealt with in an entirely different fashion. Under these conditions one wonders whether any wooden buildings could be used. If, on the other hand, the unit is to be more or less permanently based far from the active combat area, one might question the feasibility or desirability of taking care of patients suffering from subacute and chronic diseases in tents. One should consider then the more general use of light portable buildings.

The vulnerability of the hospital to attack from the air and from the sea also requires changes in design. Tents are notoriously difficult to use in a blackout and the scattering of buildings and tents will have to be on a larger scale than was possible with the space assigned. If the hospital is to be used in war conditions as a more or less permanent base the structure should be located off the main road. If it is to be

used as an active hospital for combat injuries, its position on roads will of course be different.

All in all, no one who had any experience with the Mobile Base Hospital No. 1 can doubt the advisability of having performed the experiment, nor can anyone doubt the genuine effort exerted by everyone not only to make the experiment one which would illustrate the goodness or badness of the various features but to make it a complete success. It so happened that because of the rapid expansion of the station the hospital not only served as an experiment in mobile base hospital operation, but actually was a lifesaver in taking care of the sick, owing to the shortage of beds in the dispensary and in other organizations. This might be considered as velvet in the experiment and it was certainly thoroughly appreciated by the staff officers of the station and of the Marine Corps. Undoubtedly the lessons which were learned will be considered in the erection of subsequent base hospitals of this general type and certainly some of the criticisms which I have offered will have to be discounted because of my lack of experience in this field of endeavor. The principal reason for making these observations is that it occasionally happens that one living not close to a problem will place emphasis on something which may escape the attention of those regularly concerned with the project.

THE SUPPLY OFFICER WITH A NAVAL MOBILE BASE HOSPITAL

By Ensign Phillip F. Ashler, Supply Corps, United States Navy

United States Naval Mobile Base Hospital No. 1 was experiencing no little difficulty getting much-needed supplies and provisions; its staff was beginning to realize that tropical underbrush and coral beds were taking their toll in worn clothing and broken down pedal coverings (for they could scarcely be recognized as shoes after 3 months wear in camp); the crew could never anticipate the joy of a future pay day, for they knew not when it would come. In spite of many handicaps, the unit was progressing fairly well without the presence of a Supply Corps officer, for ships in the bay were making every effort to meet the demands of the new mobile unit.

This comparative peace and serenity were destined to be short lived, for on January 19, 1941, I reported for duty as supply, disbursing, and commissary officer.

The assignment was peculiar in that, heretofore, supply officers had been assigned to other naval hospitals for disbursing duty only; the supply function being executed by a Medical Department property and accounting officer, and its commissary being directed by a pharmacist. The assignment indicated but one thing—a venture into the unknown, without the benefit of guidance by precedent.

The first unusual feature of this new mobile hospital was its organization as a unit of the forces afloat, an integral part of the United States Fleet, assigned to the command of Commander, Atlantic Squadron. Although the hospital was temporarily shore based in the Caribbean area, for accounting and administrative purposes, the unit was likened to a ship, with its home port in New York City, merely hovering at this location, ready to take flight to some unknown destination.

The first step in attempting to organize a supply department was to find a building suited to our needs; one which would provide adequate space and protection for the funds, records, and various vouchers necessary in supply work. The only location available was a wall tent—we moved in. There, with two typewriters, two desks, two chairs and a safe, we formed the nucleus of what developed to be a flourishing business. There were no forms, adding machines, or calculators available for use.

A supply of forms and the necessary equipment were requisitioned for "delivery at the earliest possible date." The rubber stamped "RUSH" on each of the earlier requisitions apparently did little toward impressing the suppliers with the urgency of our needs * * * we might have tried "FRANTIC." The forms arrived after some 6 weeks and the machines after 3 months. A limited supply of those forms essential to our carrying on was borrowed from the naval station and ships in the bay. An antiquated, surveyed hand calculator was rescued from the junk heap at the naval station and funds were transferred from the disbursing officer. With these limited facilities, and the will of innocent youth, we set out to experiment in the service of this experimental unit.

There was little difficulty involved in setting up a disbursing office. The pay accounts were transferred from the naval station to the new office, and we immediately undertook the payment of all public bills. This was accomplished on the 21st of January.

The commissary department was operating as it would on board a ship without a supply officer, functioning on a money allowance of 53 cents per day. On the first of February, it was converted to a general mess, under the supervision of the supply officer as it would be on board a vessel operating directly under the general supply system. The chief difference between the two systems is that without a supply officer, the commanding officer is allowed an amount of money for each man each day. With this he is obliged to feed the men, using his discretion as to the quantities and types of food to be consumed. The presence of a supply officer changes the picture to the extent that instead of being allowed a quantity of money, he is allowed a quantity of food stuffs. In addition, he is obliged to serve within specified percentages of meat, i. e., not less than 55 percent

beef, not more than 10 percent pork, etc. Further, of the total quantities of fresh vegetables issued during any one quarter, at least 40 percent must be Irish potatoes.

The commissary was remarkably well organized under the direction of the commanding officer, in spite of its many handicaps. The quality and quantity of food was excellent, in spite of the improvised equipment with which the men were obliged to work. The galley represented the zenith of heterogeneity as far as equipment was concerned. There were fine aluminum pots and pans; just the thing for a large family of 10; but we were trying to satisfy the hunger of 350 young men. There were no steam kettles for the preparation of vegetables, soups, and beverages. All this, in addition to the preparation of meats, had to be done in 20-gallon tin containers on the surface of 4 reconditioned, Marine Corps, wood burning, field ranges. Alongside these inadequate Boy Scout stoves stood 2 stainless steel tables, measuring 10 by 4 feet, weighing 700 pounds each, the finest in their line. To compensate for this extravagance, we were penalized with 2 hand-operated meat grinders and 2 family-size coffee mills, the sort of things one would expect to find in a backwoods general store. On the other hand, we were equipped with a fine steak cubing machine, a meat slicer and a vegetable dicer. The bake shop boasted the most modern bread slicer, but all the dough was mixed by hand and baked in out-of-door field ovens.

The staff officers were, of necessity, subsisting in the general mess. Permission had been granted the commanding officer to charge them for the meals in accordance with his actual cost of ration, i. e., although he was allowed 53 cents a day, his daily cost of ration may have been 50 cents, in that case, each officer was to be charged that amount for each day's subsistence. With the arrival of a supply officer, the Bureau of Supplies and Accounts decided that the officers should now be charged at the rate of 70 cents per day, regardless of the cost of ration. This seemed unfair in view of the fact that there would be no change in the quality or quantity of food served. The decision was based on the premise that this was a shore establishment, and as such, was entitled to a daily reimbursement of 70 cents. The attention of the Bureau was invited to the fact that although the hospital was shore based for the moment, it was a unit of the forces afloat. As such, the charge of 70 cents was not applicable. Further, it was pointed out that the officers had no alternative other than eating in the general mess, since there had been no officer's cook or steward assigned to this command. Finally the Bureau conceded that officers would be subsisted in the general mess at the actual cost of ration for the preceding month for which subsistence was furnished. Under present instructions, a daily record is maintained,

indicating the number of meals furnished each officer. At the end of the month the total is divided by 3 to determine the number of rations to be charged against the officer's account.

As the census of the hospital increased, we experienced considerable difficulty in serving ambulatory patients. Those who were bed patients were served in the wards by the pharmacist mates on duty. Those who were capable of walking about were fed in a patient mess hall. The allowed quota of messmen based on the permanent staff alone was far from sufficient to meet our increasing needs. In addition to serving the staff and patients, we were obliged to detail messmen to the galley to prepare vegetables for cooking. There were no potato peelers or vegetable cleaners furnished. In an effort to solve this problem, authority was requested from the Bureau of Navigation to include our patients in the hospital complement for the purpose of assigning and paying mess cooks. This would permit the assignment of 1 mess cook for every 20 patients as well as for every 20 members of the staff. This request was granted, solving that particular problem.

Experience demonstrated that the cold-storage facilities are far from adequate. They are not designed for tropical use nor are they designed for the stowage of sufficient quantities of meat necessary in a unit of 350 to 500 men. The meat box measures 10 by 10 by 7 feet, and seldom if ever can be brought down to a temperature of 25 degrees or less. It was estimated that a greater quantity could be stowed in the available space if the meats were purchased in commercial cuts instead of quarters. Permission to carry these commercial cuts, designed for use on small vessels, was granted by the Bureau of Supplies and Accounts. It is anticipated that we shall have an opportunity to experiment with quick-frozen boneless beef. This would permit the stowage of greater quantities of meat in the present space but would require a more dependable refrigeration system than we have been furnished.

By far the most interesting phase of development was that of supply. In setting up a system of supply accounting, it was necessary to determine which bureau or bureaus would bear the expense of non-medical supplies which heretofore had been paid out of Bureau of Medicine and Surgery allotments. It was, of course, understood that the commissioning outfit was paid for, in its entirety, by funds under the cognizance of the Medical Department. However, the question arose, whether or not this Bureau would continue to supply the necessary funds for such items as, replacement of galley and mess gear; supply office equipment; fuel and repairs to any boat assigned this unit, and other supplies and services which ordinarily would be charged to other appropriations aboard ship.

A despatch was sent to the Bureau of Medicine and Surgery, inquiring if it would continue to purchase all supplies of all classes for the unit. The reply came in a single word, "affirmative," which effectively settled all questions on this point. If that Bureau had decided otherwise, it would have been necessary for the supply officer to request allotments from the several bureaus concerned to cover the cost of such expenditures. This decision made it unnecessary to maintain the usual allotment records and accounting.

Further, if all supplies coming into this hospital were not to be charged off immediately to Medicine and Surgery, it would be necessary for the supply officer to lay in a stock of materials and charge them to the department concerned upon issue. This would necessitate sufficient safe stowage space, which at the moment is not available.

Thus we established a supply department without supplies. At no time is there a stock of any materials, other than provisions, on hand under the cognizance of the supply officer. When the property and accounting officer requests material, it is ordered from the naval station, or if it cannot be furnished locally, we prepare a requisition on the Naval Supply Depot at Norfolk, Va.

All materials received are taken up and immediately expended as a charge to the Bureau of Medicine and Surgery. The same procedure holds true with clothing and small stores. When the men are in need of clothing, a list of the desired stores is prepared and only these are drawn from the naval station or ships in the bay. At all times the inventory of any item under the control of the supply department, other than provisions, is "NONE."

This operating a supply department without supplies and storehouses works very well while the hospital is in contact with another naval activity, preferably a base. However, should a unit of this type be sent to a base where there is no local source of supply and no other naval activity, it would be necessary to provide the supply officer with adequate buildings for the stowage of materials.

To some, perhaps the explanation of our difficulties indicates that the mobile hospital would have been a smoother running unit had there not been a supply officer assigned. On the contrary, I feel that my presence here is justified by some benefits realized by this command. On the whole, it has worked for a higher morale among the enlisted force. With the presence of a disbursing officer they are now certain of regular pay days, in spite of the fact that there is little source of entertainment and recreation requiring the expenditure of money. The men are more content just to have the money in hand and spend it if the opportunity presented itself. On the other hand, there are many married men, whose families are dependent on a regular income. To these families, our presence is a guaranty of regular contributions.

In the earlier days of this unit, the commissary steward was obliged to scout about the ships in the harbor in search of fresh fruit and vegetables. The senior officer present afloat would designate one ship to furnish a boat (for most of the time we had no boat, and when we did it was seldom in running condition) and another ship to furnish provisions. There was always difficulty getting the two ships together. When there were no ships in the bay, there were no fresh vegetables on the menu.

Ofttimes it was necessary to resort to fresh-killed local beef for a meat course. To those of you who have experienced a table engagement with a steer who wore himself ragged running about the mountains, I need offer no further explanation. Now all this has been remedied. The naval station supplies have been sufficiently augmented to permit our drawing all our needs from that single source. Should we move to another location where there is no evident supply, the Commander, Train, Atlantic Fleet will arrange for a "Lady Bountiful."

Having been informed that a supply officer was ordered to the unit, the Post Office Department permitted the sale of money orders by the navy mail clerk. However, the clerk was obliged to transport his daily collections to the station where the disbursing officer would issue a check in exchange for his cash to make possible the required remittance. This meant that a considerable amount of the clerk's time was taken up with travelling rather than selling money orders. Not infrequently did it require 3 days of selling to satisfy the demands of the crew after an infrequent pay day. This presented the danger of loss of precious dollars and time to the men. This has all been corrected. The mail orderly now remains open for business until the last man has made his purchase. His travel time has been reduced to stepping from one office in the camp to another.

The presence of a Supply Corps officer has facilitated the transfer of materials from ships in the harbor; it has permitted immediate open purchase of required materials without delay; it has facilitated the drawing of much-needed clothing for the men; it has established definite sources of supply for provisions and materials; and it has furnished the doctors with an opportunity to learn about the operations of another branch of the service.

I sincerely hope that the commanding officers of future mobile hospitals and those engaged in their administration may benefit from our experiences. So many of the problems that presented themselves could have been so easily solved when the idea for this unit was conceived, had there been someone present who was familiar with the nature of the difficulties that were likely to arise. Few errors in judgment were made when the x-ray equipment was ordered, when the operating room was fitted out, and when the sanitation system

was planned. Unfortunately, we cannot say the same for matters concerning supply, disbursing and commissary equipment. I believe this can be corrected if a Supply Corps officer is ordered to a mobile base hospital at its inception.

UNITED STATES NAVAL MOBILE BASE HOSPITAL NO. 1

FROM THE VIEWPOINT OF A CIVIL ENGINEER

By Lieutenant, junior grade, Wayne D. Schoonover, Civil Engineer Corps, United States Naval Reserve

It is logical to assume that the first thing to be defined clearly in the creation of any unit is the purpose of its operation. After this is done a complete study of the problems involved leads to the development of plans and specifications, from which the unit becomes an entity. Without recourse to such a procedure the development of any unit results in confusion and waste and, what is most important, the desired end is not so likely to be obtained. There may be times when "any means justifies the end," but this is dangerous philosophy when applied to creation.

When confronted with a problem to be solved, the engineer is no different than any other person in his curiosity regarding purpose and result. It is only in the application of his special technical training to planning and development that he may excel. You go to a surgeon for an operation; you go to an engineer for a plan.

Being the first unit of its kind, United States Naval Mobile Base Hospital No. 1, may be defined as an experiment. Now, an experiment is scientifically "the arrangement of the elements or essential features of some object or process so as to permit controlled observation with a view to test some hypothesis of theory." This does not mean that an experiment is conducted or performed without thought and planning, for that would be the surest way to defeat its purpose. It does mean that an experiment, to be successful, should be conducted with forethought and careful planning along consistent lines by the application of known facts. Even when this is done, and the experiment is termed a success, it is not meant that perfection has been obtained. It may be necessary to conduct further experiments, applying the knowledge that was acquired from the first one. Thus, the mobile base hospitals that follow may be additional experiments, but should more nearly approach the desired ultimate for units of this type. It is to be hoped that on future units there will be greater coordination between conception, design, specification, and purchase.

It is not entirely correct to use the word "mobile" in naming the United States Naval Mobile Base Hospital No. 1. The word "transportable" might have been more appropriate as the unit actu-

ally lends itself to portability instead of mobility. In other words, the hospital may be carried from place to place, but it cannot move under its own power. This affects the planning considerably, as it involves the problem of suitable transports or carriers. Ships, railroad cars, and trucks all might be used for transportation of these units. Therefore, all equipment and material must be crated, packaged, or bundled in such a manner so as to be easily loaded and stowed in all classes of carriers.

One of the major problems connected with this hospital was the transportation of all material and equipment from point of origin to final destination. This involved several factors, namely, (1) packing and crating, (2) assembling and storing, (3) loading and stowing, (4) unloading and hauling, (5) storage at site or receiving point, and (6) segregation of materials. Care must be taken that equipment and material needed first at the site will not come out last. Judgment must be exercised regarding size and weight of individual items in order that handling facilities will not be overtaxed. Perishable goods must be protected at all times. All material must be classified and segregated by consistent methods in order to permit rapid and proper distribution. All these factors must be coordinated in proper sequence for satisfactory delivery of all materials to the final location.

It was intended for this hospital to be self-sufficient. This meant that during the construction period, and after the unit was in operation, no outside aid in men or materials could be expected. Of course, the usual general supplies such as food and fuel were furnished by others at regular intervals. The raw-water supply also was supplied from outside sources. Construction equipment, tools, and electric power all were included as part of the unit. Labor was furnished by the hospital personnel. Problems of housing, feeding, and disposal of wastes were of primary importance and had to be solved quickly. Shortages of various items of equipment and material were so common that if an outside source of supply had not been available the situation would have been serious. This would indicate the necessity for more careful planning. A unit must be complete in every detail to be entirely self-sufficient.

The lack of adequate construction equipment and the deficiency of building materials, such as pipe, wire, and electrical fittings, were the cause of much delay in the building schedule. For example, shortage of pipe and fittings delayed the completion of the cold-storage plant 3 months. It should have been in operation within the first 2 weeks. All heavy pieces of equipment had to be handled by hand, using ordinary trucks, whereas one small crane would have saved many hours of slow hard labor.

A regular construction schedule should be planned for future hospitals of this type. Methods of transportation, equipment, local con-

ditions, and type of personnel all affect the procedure. The units should be erected either by advance crews or by local construction forces. The use of experienced personnel with proper and adequate equipment would effect a rapid and efficient completion of the hospital.

HUMAN PLASMA AND SERUM

DEVELOPMENT AND CLINICAL INDICATIONS

By Lieutenant Commander L. R. Newhouser, Medical Corps, United States Navy, and Captain Douglas B. Kendrick, Medical Corps, United States Army

Blood plasma and serum are the therapeutic agents of choice for the emergency treatment of traumatic shock, hemorrhage, burns, and other conditions in which fluid and electrolyte balance are altered.

Human plasma has been found to be the most satisfactory fluid for the replacement of lost blood volume and the restoration of depleted blood proteins; these findings have made plasma transfusions a procedure of common practice. This does not mean that blood transfusions should be or can be abandoned. On the contrary, there is a definite place in the category for both blood and plasma.

Plasma does offer certain advantages which are worthy of consideration. It can be used without typing either donor or recipient, may be stored for many months in the liquid, frozen, or dried state and then administered safely. Long distance transportation does not alter its normal constituents. It can be made available for instant use and when injected it does not tend to increase the concentration of red blood cells in cases of shock and burns when hemoconcentration is already present.

Plasma and serum differ in composition and methods of processing. Plasma is the supernatant fluid which separates from the cellular elements when an anticoagulant is added to blood. Serum is the liquid portion of blood which separates when blood clots. Plasma contains albumin, globulins, and fibrinogen. In serum the fibrinogen has been removed in the process of clotting. The relative merits of plasma and serum have provoked a great deal of controversy but it may be said that when properly prepared both serum and plasma produce results which are quite comparable.

HISTORICAL RÉSUMÉ—LIQUID PLASMA

Blood plasma as a substitute for whole blood was suggested as early as March 1918 by Gordon R. Ward (1) in England. He pointed out that one of the chief troubles with whole blood was that the recipient's plasma might hemolyze the corpuscles of the donor. He made the observation that death from hemorrhage was not due to lack of hemoglobin but from loss of fluid, and thus the great need in these patients is for the replacement of the depleted fluid. He suggested that this

could be done by the administration of citrated plasma which could be preserved easily and injected safely.

Rous and Wilson (2) in 1918, while working on hemorrhage in animals, used plasma to replace the blood loss. As a result of their experiments they pointed out that the replacement of red blood cells is not essential, because even in severe hemorrhage a sufficient number of cells remain to support life. The loss of blood volume is the important factor. Following gross hemorrhage, they were able to restore the blood pressure to normal and maintain it by replacing the removed blood with an equal quantity of plasma.

Mann (3) in 1918, discussed the use of serum for treating surgical shock produced experimentally in dogs. He noted that the parenteral injection of homologous serum produced results which were as good or even better in the treatment of experimental shock than any other method at his disposal. It is worthy of mention that his best results were obtained with relatively large doses of serum. He asserted that homologous serum might be of value under conditions where serum could be kept and whole blood could not be obtained.

Strumia (4) began using human serum intravenously in 1927 for the treatment of severe infections. Later in 1927 he began using plasma because of its simplicity of preparation and its greater yield. It was noted then that even heterologous plasma given intravenously caused no reactions, whereas homologous sera commonly caused severe reactions. This difference in behavior of plasma and serum had previously been observed and commented on by Brodie (5) in 1900. Untoward reactions from serum were thought by him to be due to the process of fibrin precipitation when serum is separated from the clotting blood. This hypothesis continues to be conjectural.

Weech, Goettsch, and Reeves (6) in 1933 while doing plasmapheresis experiments in dogs were able to produce a normal state in their animals by administering transfusions of normal dog serum.

The use of plasma intravenously in man as a hemostatic agent was reported by Filatov (7) in 1935. The same year Heinatz and Sokolow (8) used plasma in the treatment of hemolytic shock.

Elliott (9) in 1936 suggested the use of untyped serum and plasma for the treatment of surgical, obstetrical, or traumatic shock where transfusions were indicated. He rationalized that the need for replacing the lost blood volume was more important than the red blood cells, as the maintenance of osmotic pressure is a function of the plasma proteins. Elliott also propounded the idea that liquid plasma could be stored for long periods without deterioration. It is now the consensus of opinion that liquid plasma should not be stored more than nine (9) months.

Fantus (10) in 1937, advocated the use of normal human serum because of its therapeutic and natural immunizing properties. He

states that in shock without hemorrhage and in burns, that the intravenous injection of blood serum is much more rational than that of blood, because these patients usually have an excess of red blood cells per cubic millimeter.

Alovski and Burcena (11) published a favorable report in 1937 on the use of plasma as a substitute for whole blood in gynecological conditions. Mahoney (12) in 1938, recorded encouraging results with preserved plasma in the treatment of experimental and clinical shock. Elkinton (13) and McClure (14) 1939, working separately obtained good results with plasma in burn cases. Tatum, Elliott, and Nessel, (15) 1939, suggested a technic for the preparation of whole blood substitutes in war conditions. Plasma was recommended as "an ideal substitute for whole blood in the emergency treatment of shock and hemorrhage from war wounds." They outlined the technic of collecting blood in a vacuum bottle, centrifuging the blood and then aspirating the supernatant plasma into a second vacuum bottle for storage. In 1940 Elliott, Busby, and Tatum (16) recommended the use of diluted, preserved, liquid plasma. The same year Strumia (4) advocated citrated blood plasma without cross-matching for the treatment of burns and shock. Thalheimer (17) reported favorably on the use of convalescent serum for administering antibodies. Best and Solandt (18) discussing their work on experimental shock reported encouraging evidence in favor of using plasma and serum in preventing shock. Levinson, Neuwelt, and Necheles (19) have shown the value of serum in the treatment of post-hemorrhagic shock in experimental animals. Kendrick (20), 1941, advocated the use for military purpose of concentrated and normal dilutions of plasma for the prevention and treatment of shock in the combat zone. Kekwick and Whitby (21), 1941, after using it for the treatment of shock and hemorrhage in air-raid casualties in England, concluded that plasma and blood are equally effective in restoring blood volume in injuries of this type.

DEVELOPMENT OF DESICCATED PLASMA

The previous remarks have been confined to liquid plasma. With the development of drying equipment for desiccating biologicals dried plasma has become a reality. Serum was dried successfully as early as 1896 by Martin (22), who described a simple method of preserving serum by evaporating it to near dryness under a partial vacuum at 40° C. Since then Rosenau (23), Burrows and Cohn (24), Hartley (25), Edwards and Kay and Davie (26), Hartman (27), and others, have prepared dried serum and plasma by distillation or per-vaporation from the fluid state.

Shackell (28) in 1909, first described the desiccation of biological substances from the frozen state. Since that time numerous reports

on the processing of biologicals by sublimation from the frozen state have added to our knowledge of this subject. In 1935 Elser, Thomas and Steffen (29) and later Flosdorf and Mudd (30) recommended preservation of serum in the dried state. In 1938 the latter workers (31) developed the cryochem process which utilized calcium sulphate as a chemical desiccant for drying. In 1940, there appeared in the literature a description of the Desivac drying machine, also developed by Flosdorf, Stokes, and Mudd (32). In this process the moisture is removed under vacuum and carried into the oil in the vacuum pump. The moisture is removed by circulating the oil thru a De Laval type continuous centrifuge "separator." In 1940 Hill and Pfeiffer (33) described the Adtevac process for drying plasma. The efficiency of this system is dependent upon the ability of silica-gel to absorb large quantities of water vapor under vacuum.

LIQUID, FROZEN, AND DRIED PLASMA

Serum and plasma are now being prepared in the liquid, frozen, and dried forms. Properly prepared liquid material may be administered safely after 6 to 9 months storage without refrigeration. There is, however, a loss of prothrombin and complement.

Frozen plasma and serum can be preserved indefinitely without deterioration of the normal constituents if stored at -15° C. or below. Such material should be thawed at about 37° C. to prevent precipitation. When thawed at this temperature it may be stored for 6 to 8 weeks without evidence of "fibrin" formation.

When dried plasma or serum is restored to liquid plasma, pyrogen-free distilled water is used as a diluent. Dried plasma should not be restored more than 6 hours in advance of use. To prepare 4 times concentrated plasma only $\frac{1}{4}$ of the original water content is added to the dried material. Properly prepared dried plasma, i. e., free from fusion and having a moisture content of less than 1 percent, can be preserved for at least 5 years.

CLINICAL INDICATIONS

Plasma and serum are now generally accepted as adequate vehicles for the restoration of depleted blood volume and plasma protein deficiencies. The rapid strides that have been made in the development of desiccating machines has made it possible to dry relatively large quantities of serum and plasma for experimental and clinical use. The uses to which plasma and serum have been put have demonstrated their effectiveness as therapeutic agents in many different medical and surgical conditions. Hughes, Mudd and Strecker (34), state that concentrated lyophile serum is effective in reducing increased intracranial pressure. Bond and Wright (35) report good

results with lyophile serum and plasma in the treatment of hemorrhage and shock. Aldrich, Stokes and McGuiness (36), have used concentrated serum to reduce the edema in nephrosis and their findings are encouraging. The preparation and administration of dried plasma by Strumia (4) and the use of concentrated plasma by Hill (37) in burns, shock and hemorrhage have produced results which substantiate the most optimistic claims that have been made for this agent.

In nutritional edema and hypoproteinemia, plasma infusion offers a satisfactory method for helping to replace the low blood proteins. It is especially helpful in those cases where operative procedures are indicated. Other medical conditions in which plasma and serum are an adjunct in treatment are: Infections, post-operative obstruction following gastric or intestinal surgery, gastro-intestinal hemorrhage, nephrotic syndromes and some cardiac states.

It is felt that plasma and serum have their greatest use in surgical emergencies. Here the results are frequently dramatic. In shock there is an immediate need for replacement of the lost blood volume. In order to restore this volume, colloidal solutions are essential and therefore plasma or serum are the fluids of choice in this condition. This is also true of burn cases where tremendous quantities of plasma are lost. The logical therapeutic agent to restore normal physiology is plasma. It must be remembered however, that in the treatment of burns anemia may develop, making it necessary to use whole blood as well as plasma. Hemorrhage from surgical, obstetrical and traumatic causes are successfully treated by the administration of plasma. It is advisable to give large quantities of plasma for the control of gross hemorrhages. It has been shown by Kekwick and his co-workers that the best index for determining the amount of plasma to be given is blood pressure determination.

There are other surgical conditions other than emergencies which require plasma. Wound healing is benefited by maintaining the blood proteins at a normal level. Wound disruption occurs more frequently when the protein level is low and there is tissue edema. Thompson, et al., (38) have studied the effects on wound dehiscence of hypoproteinemia in dogs and found it occurred in 70 percent of their animals. They have used dried serum to correct this condition. Plasma has been used with satisfactory results in reducing post-operative pulmonary edema. It seems likely that concentrated plasma and concentrated human albumin should be of value in the treatment of certain war gas poisons.

DOSAGE OF PLASMA OR SERUM

Secondary shock.—The cardinal principle in the use of plasma or serum is to administer an adequate amount early. The more recent reports on the treatment of shock show that the injection of relatively

large quantities of plasma or serum is essential to restore normal circulating blood volume. It has been demonstrated both experimentally and clinically that approximately 25 percent (1 liter in man) of the plasma volume is lost in mild shock. In severe shock this loss may be doubled. It becomes apparent then that the injection of small amounts (250 cc. to 500 cc.) of plasma are entirely inadequate in restoring the depleted plasma volume. Kekwick et al. have shown that it is necessary to give to the severely injured 1-2 liters of plasma or serum in order to elevate the blood pressure to a physiological level. There is a direct ratio between the amount of plasma needed and the elapsed time after injury. If the plasma is administered early the quantity required is greatly reduced. Better clinical results may be expected by giving large amounts several hours after injury.

Hemorrhage.—In the emergency treatment of hemorrhage, plasma provides an agent which is readily accessible and can be given immediately. It has been pointed out by Ward, Levinson and others that death from hemorrhage is not due to lack of red blood cells but from loss of fluid volume and thus the great need in these patients is for the replacement of the depleted fluid. This loss may be replaced at once by the administration of plasma, followed later by whole blood transfusions.

Burns.—In burns the initial disturbance in physiology is similar to that seen in traumatic shock, in that large quantities of plasma are lost. Therefore the restoration of plasma proteins plays a major role in the treatment of this injury. It has been shown that a burn involving the forearm may produce a loss of 7.0 grams of protein within the first four hours. It follows then, that burns involving more extensive areas may be expected to cause a proportionately larger loss of plasma protein. In the treatment of burns involving one-sixth to one-third of the total body area it is essential that large amounts of plasma be administered. During the first 24-48 hours it may be necessary and frequently imperative that 3-6 liters of normal plasma be used. This will have to be supplemented by additional amounts of 1-2 liters daily. The amount and length of time that will be required may be ascertained by red blood cell counts, hemoglobin, hematocrit, and serum protein determinations. If laboratory facilities are not available, sufficient plasma should be given to relieve edema.

Hypoproteinemic states.—In hypoproteinemic states resulting from nutritional disturbances and organic disease such as the nephrotic syndrome and hepatic disease, the needs for plasma differ. In the former condition, resulting from operative procedures, inanition, diarrheas, and other similar states the requirements for plasma or serum may be determined by serum protein estimations and clinical

manifestations. It may be expected that clinical improvement will follow the administration of adequate quantities of plasma. In the latter conditions, on the other hand, the amount of plasma necessary is usually excessive and may provide only temporary improvement. When it is realized that in a nephrotic syndrome the patient may lose 15-35 grams of protein daily, the futility of trying to maintain a normal serum protein by the administration of plasma becomes apparent. Patients suffering from acute hepatitis are often definitely benefited by the use of plasma. The dosage may be regulated by blood chemistry studies, total proteins, albumin, globulin, fibrinogen, and prothrombin studies.

SUMMARY

Plasma and serum are now generally accepted as the fluids of choice in the treatment of shock and burns and a valuable adjunct to whole blood as an emergency measure following hemorrhage. Plasma has some definite advantages over whole blood. It is ready for instant use, does not have to be typed, can be preserved for long periods, does not have to be refrigerated except in the frozen state, can be easily transported without changes in its constituents and when administered to patients in shock it does not increase hemoconcentration.

For the reasons stated above, and because of the marked improvement shown in patients following its use, it is believed that plasma and serum should occupy a place of prime importance in the therapy of surgical and medical conditions where its use is warranted.

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PSYCHIATRY IN AVIATION¹

By Captain J. C. Adams, Medical Corps, United States Navy

Psychiatry in aviation is one of the outstanding subjects confronting the service. Its importance is not overstressed when we realize that in an analysis of our crashes due to the human element, the fault lies, in the vast majority of cases, in failure of the intricate functioning of the central nervous system. Its importance is also stressed when we realize that almost 50 percent of applicants in past years failed to successfully complete training.

Our physical standards for flying are probably the most rigid and thorough of any country in the world. We would expect, therefore, that accidents due to demonstrable physical defects would be few. In fact, they are negligible. However, we have not been able to solve that elusive factor to which we refer as an individual's fundamental personality, with all that it implies. This is variable between individuals, which means success for one and failure for another. Not only is this grossly demonstrated by the large number who are unable to successfully complete their flight instruction, but among the successful, it remains the unknown factor that may spell disaster in the subsequent years of their flying career.

Flying is distinctly an abnormal and hazardous environment. Biologically, man has no constitutional endowment, particularly from

¹ Read before the meeting of American Psychiatric Association, May 5, 1941.

an heredity standpoint, to assist him in his adjustment to flying. We are dealing with a situation rich in profound emotional stimuli, which must be regarded as additional stress to those normally encountered in ordinary life.

Recognizing the importance of this fact, we resolve our problem into two principal divisions for control. One is directed at improving the methods of selection of flying personnel. The other is directed at improved supervision and care of flying personnel. With regard to the first objective, there has been a vast amount of research and effort directed toward improving the methods of selection with regard to psychological aptitude for flying. The Navy has conducted studies along this line for over 10 years, and has compiled thousands of records. We have recently completed a careful analysis of all these records to determine the value of this endeavor. While this analysis reveals many valuable facts, in the main it shows that we have not adequately solved the problem.

This may raise many questions regarding the reasons for the difficulties. Without attempting to consume too much time in discussing pilot selection, some of the difficulties in this connection are pointed out.

The object of this phase of pilot selection is to procure personnel who are, first basically stable, and second, to select those who possess a temperamental aptitude for flying. It is recognized that the two attributes are distinct in a practical sense, and are frequently not combined in the same individual. We have been reasonably successful in eliminating the unstable type of person. Certainly the manifestly unstable are rather easily detected. But even in dealing with this phase of the problem, the element of latent instability is more difficult to deal with because of the element of degree. When it is realized that the examinee is not a subject seeking clinical assistance, but is bent on concealing any facts that may jeopardize his opportunity for enrollment, we have a difficult problem indeed. Obviously many undesirable subjects pass the most severe test and complete their flight training, only to become a subject for further consideration in the later years of their flying career. Even in a basically stable person, the question of his temperamental aptitude for flying, or his vocational aptitude, still remains an enigma. So many factors enter into this picture that it has been most difficult to establish a basic formula upon which to prophesy success or failure. Ability to coordinate, reaction time, persistence, tenacity, confidence, judgment, intelligence, and originality, are some of the qualities of significant bearing. Genuine interest in aviation and determination to fly is a most important attribute. In turn, these attributes will be subject to emotional influences under flight training. Finally, there are extraneous factors over which the student has no control; for instance, the instructor. Some instructors inspire confidence;

others not only destroy it, but their personalities may be incompatible with that of the student. The weather must also be considered. Flying is much easier in good smooth weather than in squally, bumpy, and overcast weather. Frequent interruptions in the training schedule with lay-offs due to weather, are trying on a student. Lastly, there is the element of expediency. In easy times training requirements are rigid and failures are many. In times of emergency, standards are more relaxed and the passes are greater. It is seen, therefore, that the task is far from simple, and the chief difficulty in the past has been to evolve a successful and reliable yardstick by which to measure the applicant in the face of so many variables.

It is my opinion that part of our failure in the past has been due to the fact that we have tended to approach our problem too strictly from the angle of clinical psychiatry, and have not sufficiently stressed the approach from the standpoint of vocational psychology.

This is now being done, and in the Navy there is a comprehensive program under way in this regard. In the past, the examinations were based upon the technic of personal interview. Although the examinations were conducted by trained and experienced examiners, it has been clearly demonstrated that it is almost impossible for an examiner to eliminate his own personality in conducting these critical examinations. Also, the examinations were time consuming. A minimum of 3 to 4 hours are required for an appraisal of a personality. For this reason the method is not administratively practical when we are faced with so large a number of applicants as are now being trained.

The new method under study is based upon the principal of a self-administered questionnaire. The final analysis and scoring will be accomplished by a machine. This eliminates the personality of the examiner, expedites examinations and tends to more effective standardization. The questionnaires are directed toward providing (a) biographical inventory of the individual, (b) a vocational interest pattern, and (c) a personality appraisal. The material has been carefully compiled and is extensive. The final scores will be correlated against actual success or failure under flight instruction and we hope, finally, to evolve a validation which will be of tangible assistance in the elimination of those whose expectancy for success is too poor to justify training, or additional time to successfully qualify.

Returning now to the care of the pilot; it is clear that we have a highly selected population group engaged in a hazardous vocation. These individuals are actively engaged in a profession which requires a constant vigilance and high degree of perfection in the performance of their duties. It must be clear that even among those pilots of unquestionable skill and maximum adjustment, there are always at play the latent apprehensions of danger. As Armstrong has expressed it "apprehension, anxiety and fear are simply three stages of

comprehension of danger, and it is common knowledge that each results in profound nervous and mental depletion." It is not unreasonable, therefore, to appreciate some limit in flying time beyond which the most stable person will show evidence of deterioration. This limit varies with the individual, and considerably so depending upon the nature of the flying performed. Generally speaking about 85 hours in a month is considered a generous task.

Modern military aircraft are now so advanced that actually they can exceed in performance, the capacity of the pilot. The stresses to which the pilot may be subjected are tremendous. When we take into consideration the effect of prolonged noise, vibration, glare, and extreme cold, added to the emotional stress accompanying such flights, we have an explanation for the first condition frequently to be encountered among aviators, namely, fatigue.

Fatigue, as used here, does not refer to strictly physical fatigue as normally regarded, but to that condition commonly occurring among normal healthy pilots as a result of flying stress. It is primarily neurogenic and rapidly tends to become accumulative. It is not measured in diminished capacity to do work, but rather in the diminished will to work. These individuals do not recover their forces in the course of the average rest periods. They are tired on awakening; their morale suffers; they cease to be alert, and they suffer indifference to details. In the course of time they become inefficient and dangerous in their flying. If neglected, the picture progresses to profound staleness, and in their effort to carry on in the face of a growing sense of inadequacy, the syndrome may merge into a neurosis.

These cases when detected in time promptly respond to physical rest and a reasonable relief from flying. Their basic usefulness and attitude toward flying is not permanently impaired.

In any average population group we may expect a certain incidence of neurosis. This is true among pilots and Armstrong separates them into two main groups. First, neurosis that develops in relatively healthy stable pilots and which is found only among pilots. Armstrong refers to this condition as *aeroneurosis*. The second group is the neurosis developing among relatively unstable pilots and which is more permanently disabling, and is identical with the neurosis seen in general practice. *Aeroneurosis* is a chronic functional nervous disorder characterized by gastric distress, *gastralgia*, nervous irritability, fatigue of the higher mental centers, insomnia, and increased motor activity. Emotional stress appears to be the underlying cause of the condition. Probably the most characteristic symptoms are the stomach complaints (*aviator's belly*), and the increased motor activity. The latter symptom, so especially characteristic of *aeroneurosis*, is in contrast to the sense of weakness and lassitude seen in ordinary *neurasthenia*. The emotional stress leading to these conditions may

be associated with flying or may arise from conditions in private life or the combination of the two. Frequently the original exciting cause is a severe crash, or psychic shock as when viewing a severe crash in which the remains are badly mangled, etc. The fear complex associated with the instinct of self-preservation, the need to carry on in spite of these reactions, are factors at play. Situations in the domestic life are important factors. Debt, illness, an expected confinement, separations, etc., are most important. The syndrome and its effects are an interesting development. Time will not permit a detailed discussion in this regard, but the treatment of these cases is more complicated than would first appear. Complete relief from flying and change of environment are usually required. This is further complicated by the associated anxiety at the possibility of losing flight orders and thereby suffering a reduction in pay. Recurrence of the syndrome is frequent on return to flying. Obviously early detection and treatment are essential if the pilot is to be permanently restored to flying duty.

Under conditions of war the anxiety states and situational neurosis occur more frequently. In fact, the emotional situation confronting the pilot under actual war conditions is such that the percentage of pilots effected in one degree or another is often relatively high. Hysteria, which is not common among pilots who have completed training, is more frequently encountered under training conditions, and in time of war. We have found by experience that success for all such cases is largely preventive. Aside from careful selection of personnel, this can best be accomplished by trained medical officers who have a broad and practical aviation experience and background. It is essential that these officers come to know intimately their pilots. They must live close to them and share their experiences, if they are to obtain their confidence. An experienced flight surgeon has a practical grounding in psychiatry, but more than this, he must have a practical conception of his mission. Pilots are normal individuals and they resent being spied on, "psyched," as they call it, as if they were cracked in the head, and unless the bonds of relationship between them and their medical officer are healthy and natural, they will evade him and conceal their difficulties. When a case has progressed to that point where the average clinician can see there is something wrong, it has usually gone too far for the pilot to be restored to useful flying. Detection must come much earlier and can best be accomplished by the flight surgeon, who flies with his men and understands at first hand the true dilemma of his patient.

Generally speaking, the career of a pilot may be separated into about three periods. During the first period extending over about 4 years from time of graduation, if he is reasonably adjusted he is carefree and flies with considerable abandon. He is less conserva-

tive and has few inhibitions. He then merges into the next period which extends for about 10 years, ranging from say 27 to 37. During this period he becomes more conservative. He has frequently experienced or witnessed crashes and he has developed a more practical appraisal of his profession. He usually has acquired a family and he may be regarded as being at the peak of his profession. The next period is associated with more mature years. As the pilot grows older he incurs certain physical limitations. His domestic and social responsibilities increase and he inclines more toward administrative duties and tends to lose contact with the ever increasing advancements of the improved types of aircraft. Under these circumstances he may give up flying, or restrict his flying to a narrower field of activity. If he maintains his adjustment he may continue for an indefinite period. Maladjustments often result in the assignment of a pilot to a type of flying duty for which he has lost contact. He becomes conscious of his limitations and may develop an anxiety as to his ability to cope with the situation.

The keenly intelligent and analytical type of pilot is not always the best risk for the long pull. These individuals are more capable of sensing danger and of appreciating danger than the more superficial or dull type of personality. Undoubtedly they are subject to greater emotional response under these circumstances. They respond more sensitively to noise and other stimuli encountered in flying. Therefore, they require a more profound adjustment because of these facts. Age is another factor. Young men under 25 qualify more readily under flight training than do older personnel. In fact the curve in this regard is progressive and relatively constant. The young pilots are, in general, better adapted for pursuit or combat planes than older pilots.

In conclusion, the early indications of fatigue, staleness, effort syndrome, and even aeroneurosis are often characterized by increased irritability and changes in the pilot's attitude to his work and his associates. The early picture is subclinical and that is the time the individual should be detected. Only trained medical personnel and flight surgeons, who know the pilots and know their habits and reactions can detect a change at this stage. It is this early diagnosis that we must depend on for salvage of flying personnel if they are to be saved for useful flying.

THE ROLE OF PHOTOROENTGENOGRAMS IN THE ELIMINATION OF ENDEMIC PULMONARY TUBERCULOSIS

By Commander R. E. Duncan, Medical Corps, United States Navy, and Lieutenant E. Ricen,
Medical Corps, United States Navy

The chief role of photoroentgenograms has hitherto been in the elimination of recently enlisted naval personnel with pulmonary

parenchymal lesions caused by mycobacterium tuberculosis hominis. Numerous other acquired and congenital lesions of the lungs, mediastinum, and heart have also been detected by this procedure. Another valuable use for photoroentgenograms is now apparent among the permanent personnel of the Navy. Obviously the magnitude of the problem and the expense involved, prohibits the use of mass chest surveys of all naval personnel. It is however believed that this procedure is both justified and indicated in all ships and shore station units in which one or more cases of active pulmonary tuberculosis have been routinely discovered. Chest surveys in these units would result in the early discovery as well as treatment of cases of pulmonary tuberculosis. A dual purpose would thus be achieved by this procedure. The individual's chance for ultimate recovery would be enhanced as well as eliminating pulmonary tuberculosis as an endemic source of infection. In the past, numerous examples have been noted in which multiple cases of pulmonary tuberculosis from a particular unit have been admitted to a United States naval hospital during the course of a year. The value of prompt chest surveys of naval personnel in such units is incalculable. The only logical method of eliminating pulmonary tuberculosis is attacking the problem from two directions. Already a major step has been made by the routine chest surveys of recruits. The application of this method to all individual units in which pulmonary tuberculosis has been found to exist will eventually result in the solution of the problem of tuberculosis in the Navy.

The responsibility for initiating group chest surveys lies with the medical officer attached to each particular unit falling in this category. The factor of promptness in instituting this procedure is all important and cannot be too strongly emphasized. The futility of routine physical examination of the chest as a substitute for photoroentgenograms is generally accepted as a fact and will therefore not be dwelt upon at length. Suffice it to say that unfortunately a great discrepancy exists between the physical findings elicited by examining the chest and the underlying pathology. It is not unusual for minimal or even moderately advanced tuberculous lesions to present so slight a variation from the normal chest as to be undetectable by the stethoscope. To illustrate the value of routine chest surveys for naval units in which pulmonary tuberculosis has been proven to exist the following case reports are cited:

CASE REPORTS

E. W. B. M. 2c., admitted to the United States Naval Hospital, Washington, D. C., August 20, 1940, from the Navy Yard, Washington, D. C. Chest film on admission revealed a diffuse, soft, and sketchy infiltration throughout both upper lobes. There is a cavity 3 cm. in size in the right infraclavicular region. (Fig.

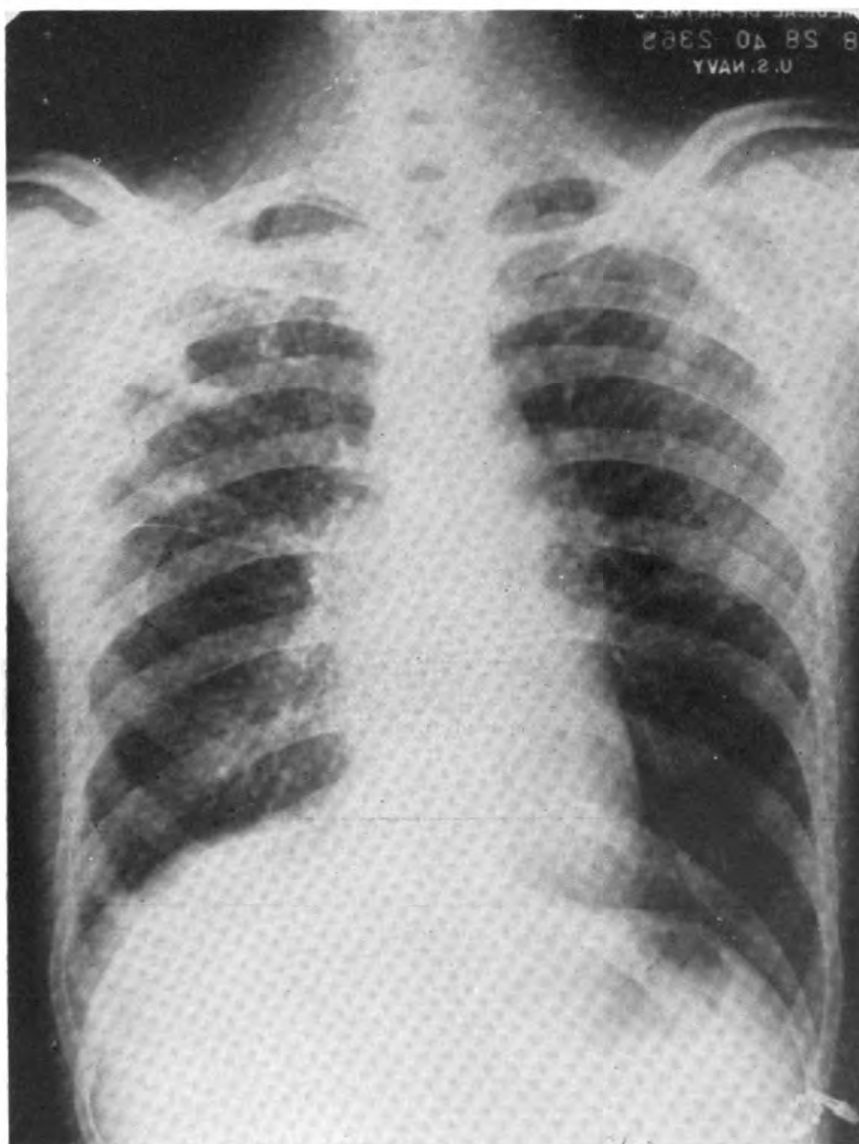


FIGURE 1.--CAVITY IN RIGHT INFRACLAVICULAR REGION.

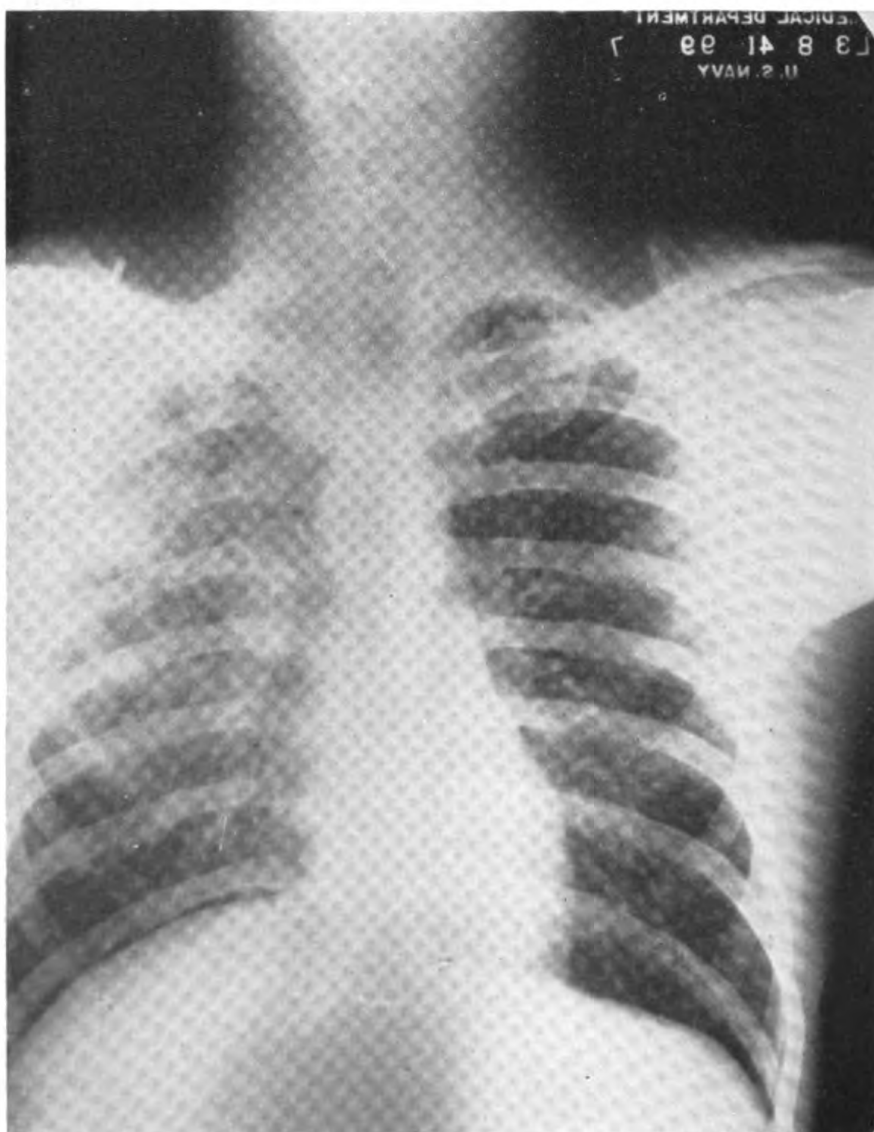


FIGURE 2.—DIFFUSE INFILTRATION IN RIGHT UPPER LOBE.

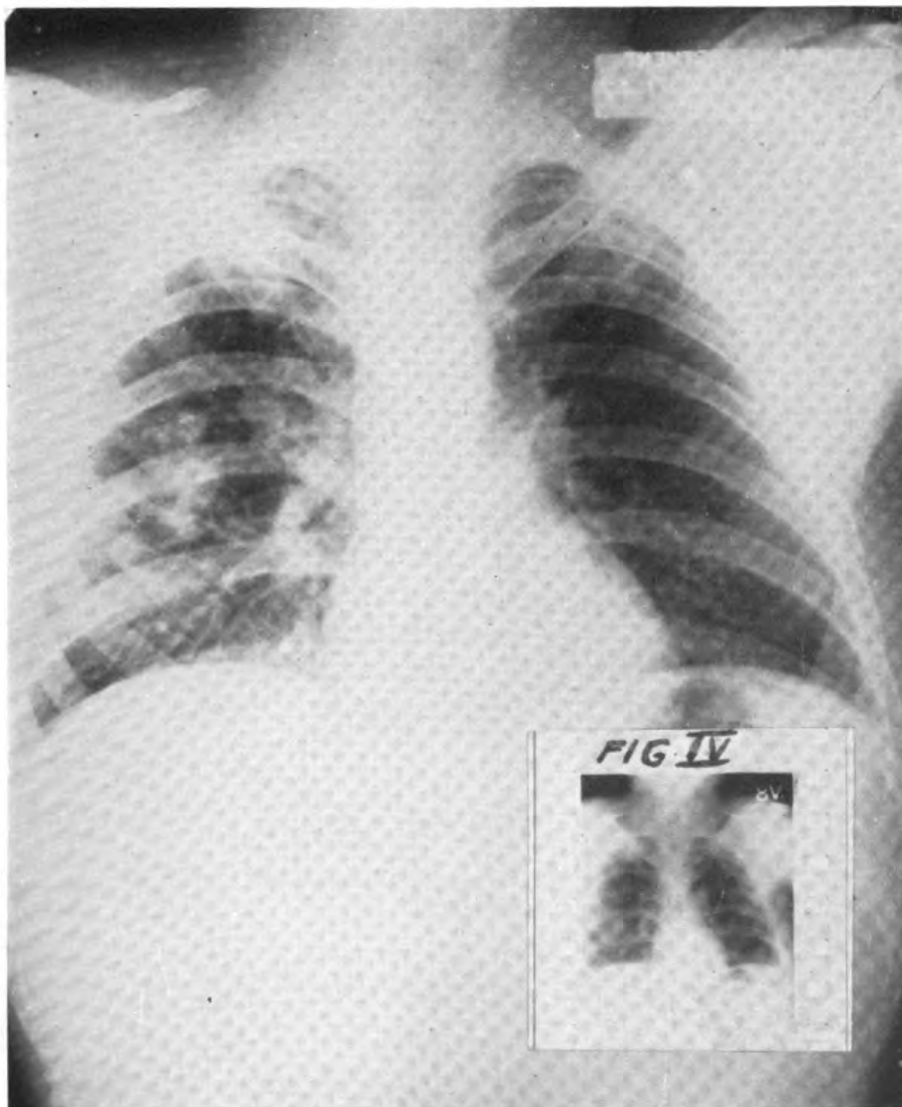


FIGURE 3.—INFILTRATION OF RIGHT APEX.
FIGURE 4.—A CONTACT PRINT FROM 35 MM. FILM TO SHOW COMPARISON.

1) Sputum examination August 30, 1940, was positive for mycobacterium tuberculosis homonis.

R. E. H. Sea. 1c., admitted to United States Naval Hospital, Washington, D. C., March 8, 1941, from the Navy Yard, Washington, D. C. Chest film on admission revealed a diffuse infiltration of woolly type throughout the entire right upper lobe. The lungs are otherwise clear except for some minimal scarring at the left apex. (Fig. 2) Sputum examination March 10, 1941, was positive for mycobacterium tuberculosis homonis.

The two cases of pulmonary tuberculosis described above were not only discovered in the same shore establishment but were further found to have been housed in the same barracks. In addition intimate association between all individuals in this barracks existed such as drinking from the same coffee cup, sharing cigarettes, etc.

In view of these facts a chest survey of the personnel attached to the barracks in question was initiated. Photoroentgenograms were run on 80 men. As a result of this survey an additional case of pulmonary tuberculosis was discovered.

E. E. Sea. 1c. Chest film reveals some irregular haziness of the right apex along with indications of soft infiltration largely concealed by the clavicle and first rib. Soft infiltration is also noticed in the lung fields several centimeters from the right hilum and also in the paravertebral portion of the right upper lobe. In the left infraclavicular region a small amount of soft sketchy infiltration is also noted. (Fig. 3) A contact print from 35 mm. film (Fig. 4) is shown for comparison.

CONCLUSION

The utilization of photoroentgenograms for conducting chest surveys of naval personnel in those units in which pulmonary tuberculosis has been found to exist constitutes the only real means of combating the problem of pulmonary tuberculosis in the Navy.

PERITENDINITIS CALCAREA

WITH PARTICULAR REFERENCE TO CALCIFICATION IN THE SUPRASPINATUS TENDON

By Lieutenant Commander William E. Pinner, Medical Corps, United States Navy, and
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The term "peritendinitis calcarea" has frequently been used by European writers to denote abnormal calcific deposits in and about the tendons of the muscles of the shoulder, hips, elbows, and other joints. These calcific shadows were first described by Painter, an American, who regarded them as having arisen in a thickened bursa. The American authors, until 2 decades ago, held to the view that bursitis was the cause of the calcification, until Brickner (1) of Chicago in 1917, reported that the calcareous deposits were located in the supraspinatus tendon and not primarily in the subacromial bursa. Carnett (2) of Philadelphia in 1929, in reporting on 50 cases of calcareous deposits observed at operation, saw no pathological changes in the subacromial bursa in the majority of instances. The one constant

lesion was the calcareous deposit situated beneath the bursa on, in, or underneath the supraspinatus tendon.

Similar shadows have been observed close to joints other than the shoulder. Sandstrom (3) found that among 329 cases observed the location of the disease was in the neighborhood of the shoulder joint in 259 cases, the elbow joint in 6, the wrist in 2, the fingers in 6, the hip joint in 48, the knee joint in 5, and the toes in 3 cases. He recognized the general nature of the disease and proposed, in 1929, the term "peritendinitis calcarea"—a name which will probably remain appended to the disease until a better one is found.

The purpose of this report is to present data showing that peritendinitis calcarea is a common disease of middle life, and that roentgen therapy directed to the diseased area is frequently effectual in relieving the symptoms and decreasing the quantity of the calcareous deposits in and about the tendons.

ETIOLOGY

Trauma seems to be the factor which precipitates the disease, but antecedent degenerative changes in and about the tendon appear to be the more important factor in the causation of the disease.

ANATOMY

There are two groups of muscles controlling motion of the shoulder. The outer group consists of the deltoid, the pectoralis major, the latissimus dorsi, and the teres major. These are all inserted into the shaft of the humerus below the surgical neck. The inner group of short rotators—the supraspinatus, infraspinatus, teres minor, and the subscapularis—is intimately associated with the capsule of the shoulder joint and forms a muscle cap over the front, top, and the back of the head of the humerus. The space between the acromium and the deltoid above, and the short rotators below, is lined with synovial membrane, this is the so-called subacromial bursa.

The supraspinatus muscle inserts on the top and the anterior slope of the greater tuberosity of the humerus. The infraspinatus inserts on the posterior slope of the tuberosity. The teres minor is inserted on the posterior edge of the greater tuberosity below the backward slope. The subscapularis is inserted on the front of the lesser tuberosity.

MORBID ANATOMY

Case, (2) working in association with Carnett, found in 26 cases that the sequence of changes in the tendon appeared to be "a primary interference with the blood supply, then ischemia and resultant necrosis." Calcium salts later were deposited in the necrotic tissue.

In many instances, but not all, an inflammatory reaction was excited. The deposits varied from microscopic to collections of considerable size. The material was soft and cheesy, resembling caseation necrosis; sometimes it was fluid or semifluid like pus. In other cases it was found brittle. Culture showed it to be sterile. Chemical analysis revealed that the deposits consisted of some salt, of calcium, of phosphates, oxylate or carbonate. Only one case showed bone and cartilage. Hemosiderin was found only twice. The inflammatory reaction varied in degree from extensive formation of granulation tissue to the scattered infiltration of a few pus cells. Only a few polymorphonuclear leukocytes were noted. The lymphocytes, plasma cells, and large mononuclear cells were the ones mostly concerned in the chronic inflammatory reaction. In many instances, foreign body giant cells were noted.

CLINICAL ASPECTS

In the acute form pain may be intense and agonizing. It may extend from the neck to the fingertips or it may be restricted to a small area in the arm at the point of the shoulder. Frequently the victim supports the flexed forearm with the opposite hand in order to restrict motion in the involved joint, and thereby lessens the intensity of the pain.

There may be localized edema in the severe case, but none in the mild case.

Tenderness is most frequent and intense in the region of the greater tuberosity of the humerus. The area beneath the edge of the acromion process is another frequent site of marked tenderness. Seldom is the spot larger than $2\frac{1}{2}$ cm. in diameter.

Motility is restricted, particularly the active movements. In the acute form the passive movements may also be greatly lessened. Raising the arm outward and upward is usually painless at the start if done slowly, but at an arc of 45° – 90° the pain is most severe in calcification of the supraspinatus tendon. Beyond the 90° elevation the pain is not severe because the lesion has passed under the clavicle and is free from pressure and tension.

Most of the acute cases lose their severe pain in from 3 to 5 weeks without medical treatment and then may go on to a rapid and complete recovery; but many pass on into the chronic form and have milder symptoms for months. These latter patients may have acute exacerbations, usually after mild injury to the joint. The latent or chronic forms are important and are often incidentally found on the roentgenograms, usually in patients referred for examination of the joints for arthritic changes.

The clinical diagnosis of this disease is not difficult. The distribution of the pain, the limitation of shoulder motion, and the sharply

localized area of tenderness are distinctive. In the clinical differential diagnosis arthritis, neuralgia, and neuritis must be considered. Surgical conditions, such as, cervical rib, avulsion fracture of the greater tuberosity of the humerus, and spontaneous dislocation of the shoulder, and sprain must also be considered.

ROENTGEN DIAGNOSIS

The characteristic feature of this disease is the calcification in the tendons and, less frequently and extensively, in the articular capsules and the bursae. Often the lesions may be apparent on the ordinary roentgenogram taken for bone detail. In many cases, however, a special technic is required to visualize the deposits in the soft tissues. The so-called "soft tissue" detail roentgenogram will reveal faint, amorphous deposits that would not show on ordinary roentgenograms. Tangential projections are required near the shoulder joint to throw the calcareous deposits away from the bones.

Blackett and Healy, (4) after working with various angles, concluded that four positions are necessary for complete study of the shoulder.

The first position is the usual anteroposterior projection with the patient lying on his back and the arm in external rotation. The central ray is directed through the shoulder perpendicularly to the film. This shows the supraspinatus in profile on the top of the greater tuberosity.

The second position is similar to the first but the central ray enters the shoulder from above at an angle of 25° with the perpendicular. The central ray is parallel with the backward slope of the posterior part of the tuberosity where the infraspinatus tendon is inserted. Codman (5) frequently employed this angle projection.

The third position shows the insertion of the teres minor. The patient lies on his face, the arm in internal rotation. The central ray is directed slightly outward at the point of the shoulder.

The fourth position employs the vertical view. The patient lies on his back with the arm abducted to a right angle in external rotation. The film is above the shoulder and the central ray passes up through the axilla. The insertion of the subscapularis is seen on the anterior edge of the humerus, and the insertion of the teres minor on the posterior edge.

In the acute cases the amorphous deposits are frequently observed and they are difficult to demarcate from the neighboring soft tissues. In the chronic cases the shadows are frequently dense and sharply demarcated. In many patients a combination of both types is present. The lesions may vary from the minute, punctate calcifications to large, amorphous collections which have no distinctive internal structure.

There is, however, a tendency for the deposition of the salts to take place along the plane with the tendon. Rarely is there evidence of trabeculation due to osseous change.

TABLE I.—*Location of the calcareous deposits in 56 cases*

		Cases
Right shoulder	Supraspinatus tendon	35
	Long head of triceps	2
Left shoulder	Supraspinatus tendon	18
	Long head of triceps	1
Right elbow	Insertion triceps tendon	5
Left elbow	do	1
Right hip	Gluteus medius tendon	5
Left hip	do	1

ROENTGEN DIFFERENTIAL DIAGNOSIS

The following diseases may at times enter into the differential diagnosis:

(1) *Calcinosis interstitialis universalis*.—refers to deposits of calcium salts in the skin and subcutaneous tissues. The etiology is unknown. There are multiple calcium deposits in the skin, subcutaneous tissues, and deep-seated interstitial connective tissues. These patients usually have had long standing scleroderma, peripheral vascular or Raynaud's disease. The early symptoms are often soreness or tenderness of localized areas of skin. The lesion is characteristically developed in areas where trauma may occur. Chamberlain (6) has found, by roentgen examination, calcified areas varying in size usually located near the large joints. They may be at different depths either in the subcutaneous areas or the deepest interstitial tissues. They are not connected with bone.

(2) *Calcinosis interstitialis localizata*—(*lime gout*).—Schinz (7) prefers to classify the punctate, localized form of calcification under the above heading. The deposits are usually located in the subcutaneous tissues of the fingertips. There is no alteration in the structure of the neighboring bone in lime gout, whereas in true gout the sodium monourate tophus occupies the punched-out area of atrophic loss of bone substance near the joint margin. The blood chemistry values are within normal limits in lime gout, whereas blood uric acid values higher than 4.5 mgm. per 100 cc. of blood serum are typical of true gout.

(3) *Ossifying myositis* is usually due to post-traumatic calcification of stagnant fluid, blood, or lymph in the muscle tissue. The calcification is usually in the direction of the muscle fibers.

(4) *Myositis ossificans progressivae*—has the same general appearance as ossifying myositis, excepting that the deposits are osseous in character due to the longer duration of the disease.

(5) *Hyperparathyroidism*.—There are often calcareous deposits in the soft tissues in this disease, but cystic lesions and decalcification of bone are not a part of peritendinitis calcarea.

(6) *Parasitic causes* of calcification due to cysticercus cellulosae or guinea worm infestation are unknown in this geographical location.

(7) *Senile hypertrophic changes* (osteophytes) often represent formation of bone from the margins of the articular surfaces and are seldom confused with the calcific deposits of peritendinitis calcarea, which are close to but not a part of the bone.

TREATMENT

In 1938, Sandstrom reported excellent results from roentgen irradiation. The technic employed was 200 kilovolts, 6 milliamperes, 40 centimeter distance, $\frac{1}{2}$ millimeter of copper and 1 millimeter of aluminum—filter. Fields varied somewhat with the type of case, but were usually 10 by 10 centimeters. In the acute form he gave from 70 to 100 roentgens every other day or every third day until the acute symptoms subsided. Seldom were more than 5 doses required in the acute cases to eliminate the pain and to restore the joint to normal usefulness. In the chronic cases he gave a series consisting of 3 treatments of 100 roentgens each, followed by an identical series after an interval of 1 month. Thereafter the interval between series was increased to 2 or 3 months, but seldom were more than 4 series required in any case.

REVIEW OF 56 CASES

There were 56 patients in the United States Naval Hospital, Philadelphia, Pa., during the year ending June 30, 1940, who showed calcification in and around the tendons in the neighborhood of at least 1 or more of the joints. Fifty-one of these were in-patients, and 5 were out-patients.

In each group of 147 males admitted to this hospital there was 1 patient who showed roentgenographic evidence of the disease.

The average age was 49.1 years. The youngest patient was 38 years old.

For convenience of reference, the size of the calcareous deposits was arbitrarily divided into 4 groups. The measurements were limited to the height and width of the opacities.

Grade I denotes lesions from the minute to $\frac{1}{2}$ centimeter in diameter.

Grade II denotes lesions from $\frac{1}{2}$ to 1 centimeter in diameter.

Grade III denotes lesions from 1 to 2 centimeters in diameter.

Grade IV denotes lesions larger than 2 centimeters in the greater diameter.

The radiographic densities varied from the poorly defined, faint, homogeneous or amorphous calcifications to the dense, well-defined and well-circumscribed opacities. Frequently, a combination of the 2 types was observed.

TABLE II.—Details

Case No.	Initials	Symptoms	Tender-ness	Movements re-stricted	Roentgen findings	Grade
1	M. L.	Chronic	I	I	Supraspinatus, left	I
2	G. M.	do	II	II	do	I
3	E. L.	Subacute	II	II	Supraspinatus, right	III
4	H. L.	do	II	II	do	I
5	J. E.	Chronic	I	I	do	II
6	G. M.	Subacute	II	II	do	II
7	F. K.	Acute	III	II	do	III
8	H. C.	Chronic	I	I	Supraspinatus, left	I
9	W. L.	Subacute	II	II	Supraspinatus, right	II
10	N. M.	Acute	IV	III	Supraspinatus, left; also, sub-acromial bursa.	II
11	W. W.	Chronic	I	I	Supraspinatus, right	I
12	J. R.	do	I	I	do	I
13	J. N.	do	I	I	do	I
14	G. P.	Subacute	II	I	Triceps insertion, left	I
15	C. S.	do	II	II	Supraspinatus, left	III
		Chronic	I	I	Supraspinatus, right	II
16	E. H.	(?)	(?)	(?)	Triceps insertion, left	I
17	J. G.	(?)	(?)	(?)	Supraspinatus, right	III
18	M. M.	Subacute	II	II	Triceps, long head, right	III
19	A. V.	Chronic	I	0	Supraspinatus, left	II
					Supraspinatus, right	I
20	G. R.	Subacute	II	II	do	III
					Subacromial, right	III
					Subdeltoid, right	III
					Gluteus medius, right	II
21	N. L.	Subacute, right	II	II	Supraspinatus, right	II
		Chronic, left	I	I	Supraspinatus, left	I
22	P. D.	Acute	III	III	Supraspinatus, right	IV
23	E. F.	(?)	(?)	(?)	do	III
24	R. C.	Subacute	II	II	do	II
25	J. M.	do	II	II	do	III
26	F. Z.	do	II	II	Triceps, long head, origin, right.	II
27	J. G.	(?)	(?)	(?)	Supraspinatus, right	II
28	G. L.	(?)	(?)	(?)	do	III
					Gluteus medius, right	III
					Gluteus medius, left	III
29	F. S.	Subacute	III	III	Supraspinatus, right	II
		do	II	II	Supraspinatus, left	II
		Chronic	I	I	Triceps, long head, left	I
30	P. McN	Subacute	II	II	Supraspinatus, left	II
31	M. J.	Chronic	I	I	do	I
32	B. W.	(?)	(?)	(?)	Triceps insertion, left	I
33	H. B.	Subacute	II	II	Supraspinatus, right	II
34	J. G.	(?)	(?)	(?)	Gluteus medius, right	I
35	S. Z.	Subacute	II	II	do	II
36	W. McN	(?)	(?)	(?)	Supraspinatus, left	II
37	V. V.	Subacute	III	II	Supraspinatus, right	III
					Triceps insertion, left	I
38	J. M.	do	II	II	Gluteus medius, right	I
					Supraspinatus, left	I
39	V. D.	do	II	II	Supraspinatus, right	II
40	B. D.	Chronic	II	II	do	II
41	M. M.	Acute	IV	III	Supraspinatus, left	III
		Chronic	I	I	Supraspinatus, right	I
42	E. S.	do	I	I	do	I
43	N. W.	(?)	(?)	(?)	Supraspinatus, left	I
44	F. S.	Chronic	I	I	Supraspinatus, right	I
45	I. F.	Subacute	II	II	do	II
		Chronic	I	I	Supraspinatus, left	I
46	B. S.	Acute	IV	III	do	III
47	R. B.	(?)	(?)	(?)	do	III
48	J. K.	(?)	(?)	(?)	Supraspinatus, right	I
49	H. S.	(?)	(?)	(?)	do	I
50	B. S.	Subacute	II	II	do	II
51	J. P.	(?)	(?)	(?)	Supraspinatus, left	II
52	E. C.	Subacute	II	II	do	II
53	S. M.	Chronic	I	I	Supraspinatus, right	I
54	N. B.	Subacute	II	II	do	II
55	P. O.	(?)	(?)	(?)	do	II
56	J. M.	(?)	(?)	(?)	do	II

We have learned to anticipate a high degree of absorption of the amorphous type of calcification after the first series and a lesser degree after the second, but we have also learned by experience that the dense, well-defined, and perhaps encapsulated lesions show very little change in size after repeated courses of roentgen irradiation. We, at present, prefer to give one course of 300 roentgens and to repeat it later when the symptoms warrant further treatment.

TABLE V.—CHRONIC FORMS

Case No.	Size of calcification prior to irradiation	After first series	After second series	Results	
				Clinical	Roentgenographic
1	I	O	—	Excellent.....	Excellent.
2	I	O	O	Good.....	Do.
8	I	N	—	do.....	N.
11	I	Trace	—	Very good.....	Good.
12	I	O	—	Excellent.....	Excellent.
13	II	N	—	Good.....	N.
19	I	Trace	O	Very good.....	Good.
31	I	Trace	—	do.....	Do.
40	II	I	I	Good.....	Fair.
42	I	O	—	Excellent.....	Excellent.
44	I	N	—	Good.....	N.
53	I	N	—	Very good.....	N.

N refers to no check roentgenogram.

— refers to no further series of irradiations.

O refers to complete disappearance of the calcareous deposit on the check roentgenogram one month after a series was initiated.

The 12 patients with mild manifestations of the disease reacted very satisfactorily to 1 course of 300 roentgen units.

The calcareous deposits were completely eliminated in 4 cases. Four patients showed minute calcifications after the first course; and 4 failed to return for the check radiographic examination.

The greater number of patients in this group was referred to the x-ray department for roentgenograms to demonstrate the presence or the absence of arthritic changes. The calcifications in and around the tendons were incidental findings. Arthritic changes were seldom demonstrable in the shoulder joints.

THE THERAPEUTIC ACTION OF ROENTGEN RAYS

The therapeutic action of the roentgen rays depends upon the well-known analgesic effect and the liberation of endotoxins from the destroyed lymphocytes, and, to a lesser degree, the neutrophils. Hyperemia and phagocytosis are believed to be increased. The avascularity of the tissues from the preceding degenerative changes is reversed and absorption of the calcium salts is hastened. The hyperemia may also cure the underlying tendinitis.



FIGURE 1.—CASE 20.—SUBACUTE FORM. PRIOR TO ROENTGEN THERAPY.

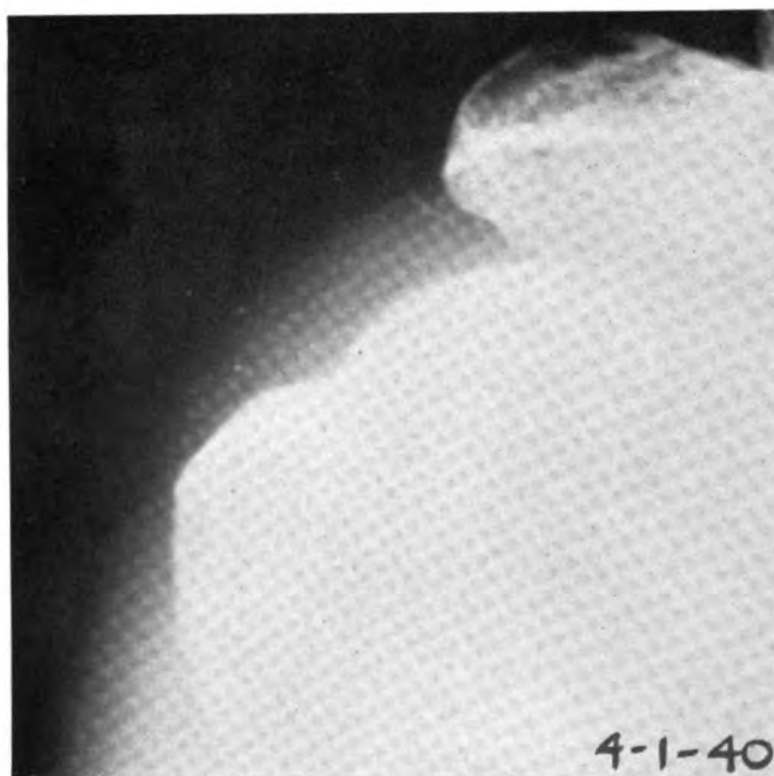


FIGURE 2.—CASE 20.—TWO WEEKS AFTER THE SECOND COURSE OF 300 ROENTGENS.
THE CALCAREOUS DEPOSITS ARE NO LONGER PRESENT

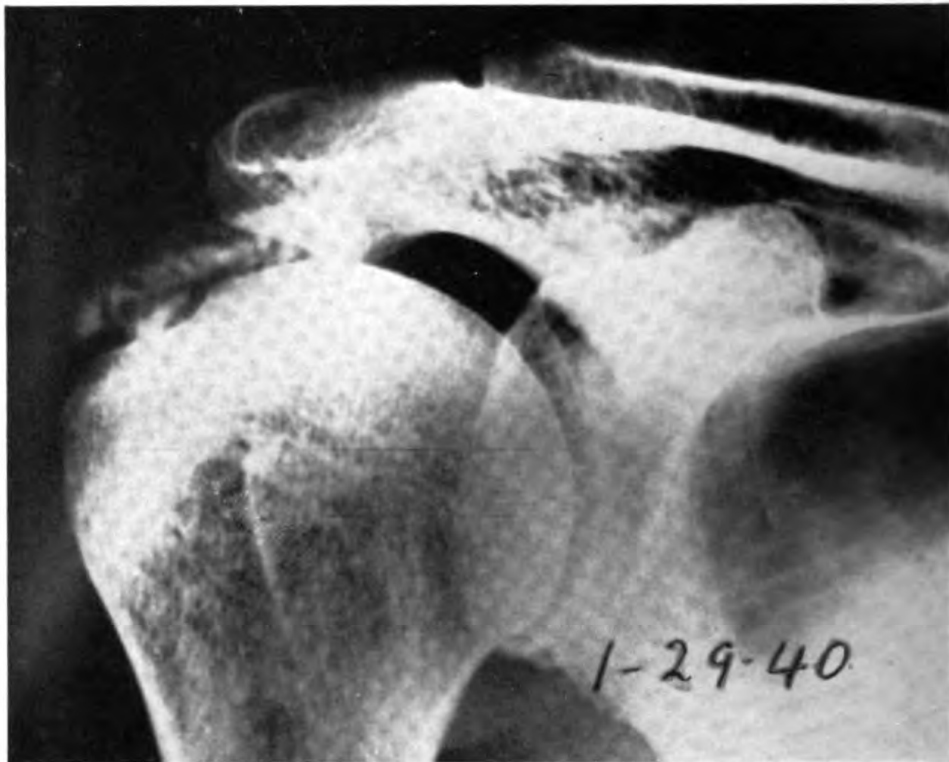


FIGURE 1.—CASE 22.—PRIOR TO ROENTGEN THERAPY

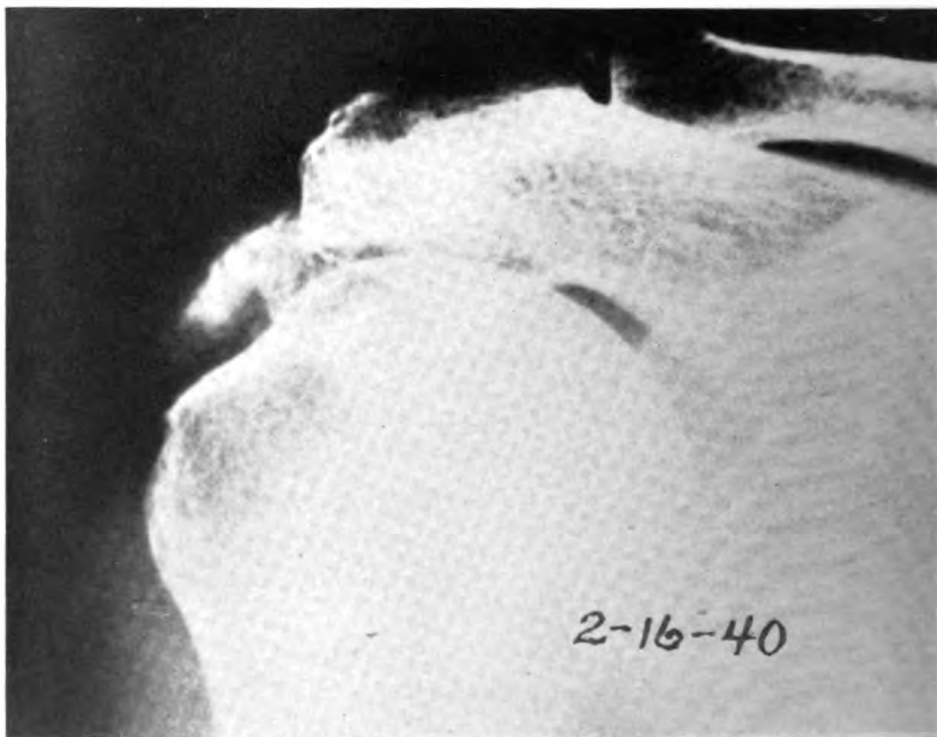


FIGURE 2.—CASE 22.—AFTER 5 DOSES OF 100 ROENTGENS EACH.

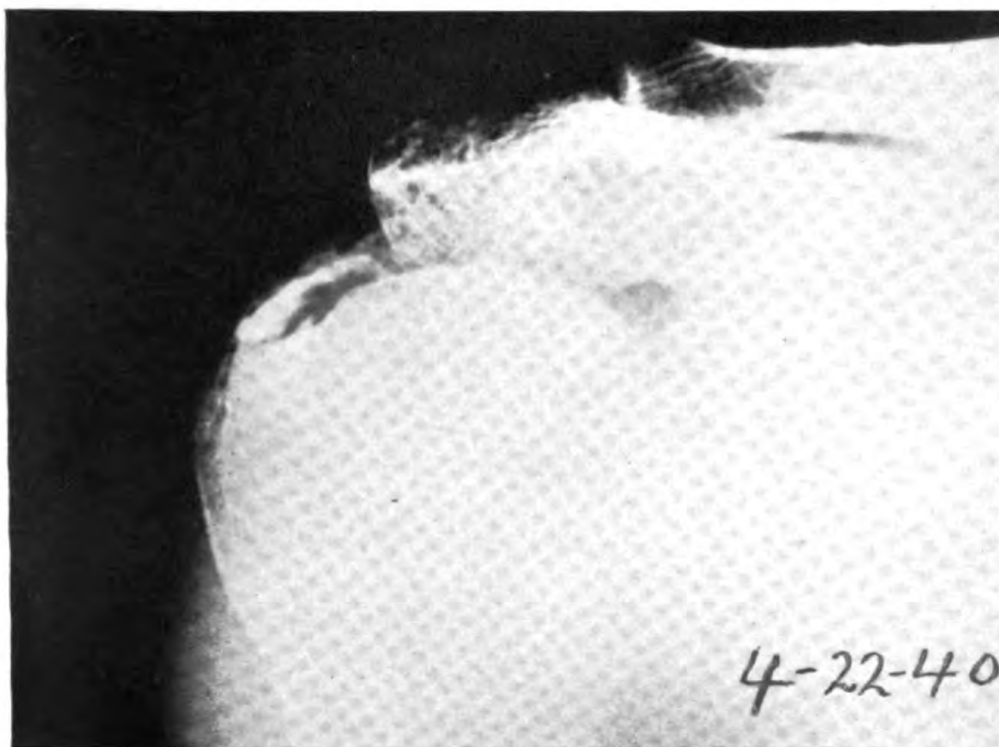


FIGURE 3.—CASE 22.—ONE MONTH AFTER A SECOND COURSE OF 300 ROENTGEN UNITS.



FIGURE 4.—CASE 22.—ONE MONTH AFTER ANOTHER COURSE OF 300 ROENTGENS

SUMMARY

We feel that roentgen therapy is the most effective measure in the treatment of calcareous deposits in and about the tendons.

In all instances marked clinical improvement was obvious after the first course of roentgen therapy. These patients were, undoubtedly, the most grateful.

The rate of absorption of the calcium deposits was almost as phenomenal as the abrupt clinical improvement.

CONCLUSIONS

1. Peritendinitis calcarea is a common disease of middle life.
2. The acute and subacute forms of the disease are temporarily disabling. The chronic form is undoubtedly responsible for reduced efficiency.
3. Spontaneous absorption of the calcareous deposits may occur due to the irritative hyperemia produced by the deposits, but the reaction is slow. Diathermy, local heat, and massage are occasionally helpful but are also slow in their effects. Morphine sulphate is at times required to control the pain in the acute forms.
4. Roentgen therapy in small doses causes the pain to promptly disappear. The joint is restored to normal usefulness after a few days.
5. The amorphous deposits undergo absorption in most instances. The relatively infrequent, but dense, well-circumscribed deposits seldom completely disappear after roentgen irradiation.
6. Hospitalization is not essential. Surgery is seldom required.

ILLUSTRATIVE CASES

Case No. 20, G. R., admitted January 19, 1940.—Onset 1 month ago "with pain in the right shoulder which lasted only a day or two and then disappeared. Two months ago the pain returned and it has persisted." He holds the arm to the chest and will not move it because of pain.

The physical examination reveals the right arm abducted to the chest wall. There is slight redness and exquisite tenderness about the shoulder.

The roentgenogram (January 23, 1940) (fig. 1), revealed a grade III amorphous calcification near the site of insertion of the supraspinatus tendon. An exceptionally large portion of the deposit was in the subdeltoid bursa.

Roentgen therapy was initiated. The check roentgenogram (February 2, 1940), 4 days after the last treatment in the first series revealed the calcareous deposits greatly reduced in size. The final roentgenogram (April 1, 1940) (fig. 2), 2 weeks after the second course of 300 roentgen units to the shoulder showed no evidence of calcification in or about the tendon.

The response to roentgen therapy was excellent. The first doses of 100 roentgens resulted in considerable improvement. He was symptom free after the first course of 300 roentgens.

a possible partial dislocation of the broken head. In the face of such extensive injury, the vertical view was impracticable and the lateral view was employed. The semilateral view through the chest showed the fragment in a different relation, but the head still appeared unchanged against the posterior rim of the glenoid. Either view alone is inconclusive of dislocation. Taken together, however, a diagnosis can be definitely established. The patient was intractable and left the hospital against advice; the treatment being limited to the application of a Jones' splint. There was no follow-up.

Case 3.—V. I. N., male laborer, 46, admitted January 16, 1935, complaining of pain in his left shoulder. Ten days previously, in the outpatient department, he had an epileptiform seizure, after which he had a continuous pain in the left shoulder, aggravated by motion. Roentgen examination of the anterior-posterior view (fig. 3 A) showed a fracture of the head of the humerus apparently in line with the posterior rim of the glenoid. The vertical view (fig. 3 B) more clearly outlines the fracture of the head and the manner in which the posterior segment of the glenoid is wedged into the fractured head, locking it in dislocation. The dislocation was reduced under general anaesthesia, but due to recurrent dislocations a Nicola operation was subsequently performed.

It is evident from the foregoing discussion and the cases reported that the important factor in the diagnosis of posterior dislocation of the shoulder is a roentgenologic view which demonstrates the dislocation. The usual anterior-posterior, or post-anterior view, although at times suggestive, is insufficient. The diagnosis cannot be made with certainty on this view alone. The shoulder does not offer sufficient depth to make stereoscopic views always accurate. A lateral or vertical view, in conjunction with the ordinary anterior-posterior view, or still better an anterior-posterior stereoscopic view, will show the deformity. The simplest and best position requires at best 75° abduction of the arm from the body, with the film preferably on top of the shoulder. Jordan (8) devised an ingenious device for holding the cassette in position with the patient sitting up. Obviously, a position requiring marked abduction of the arm cannot be obtained, or even attempted, in the presence of many of the complicating fractures of the bones of the shoulder girdle. Sometimes in simple dislocations, the pain alone is sufficient to prohibit abduction. In such cases, a satisfactory lateral view through the chest can be made. The patient stands, as for a lateral chest film, with the involved shoulder next to the Bucky surface. The sound arm and shoulder are then elevated high, in order not to impair the view. This view is also valuable in fractures about the shoulder, uncomplicated by dislocation.

SUMMARY AND CONCLUSIONS

Posterior dislocation of the head of the humerus, although infrequent, occurs often enough to warrant consideration. The diagnosis depends upon roentgenological visualization of the dislocation. This can best be obtained by a vertical or lateral view, preferably the former. Either a vertical or lateral view should be obtained in all

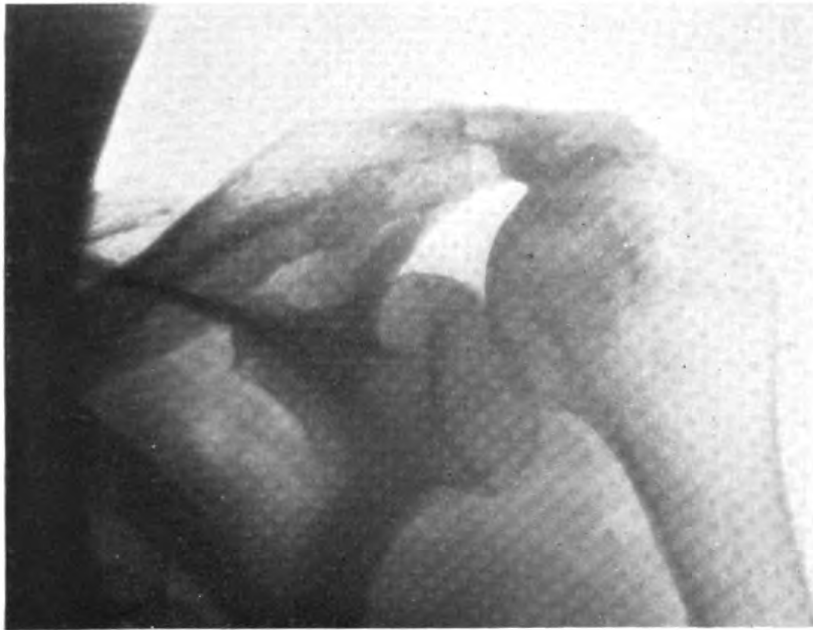


FIGURE 1A.—CASE 1.—USUAL A. P. VIEW SHOWS FEMORAL HEAD HIGH IN RELATION TO THE GLENOID AND SUGGEST THE POSSIBILITY OF DISLOCATION.

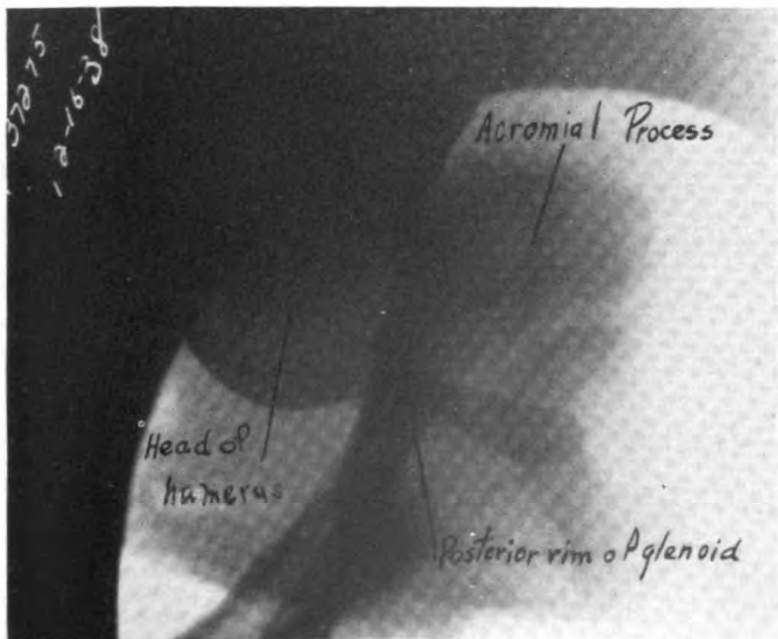


FIGURE 1B.—CASE 1.—VERTICAL VIEW SHOWS THE HEAD COMPLETELY BEHIND THE GLENOID.

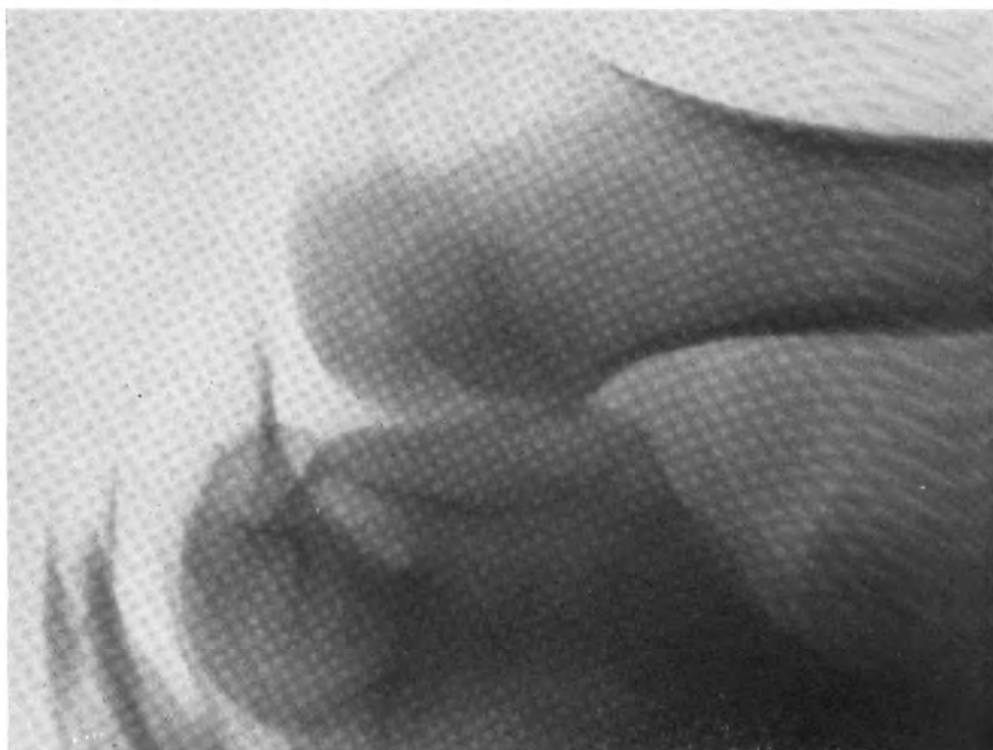


FIGURE 3A.—CASE 3.—ROUTINE A. P. VIEW SHOWS A DEFECT IN CONTOUR OF HEAD, APPARENTLY IN LINE WITH THE POSTERIOR RIM OF THE GLENOID, SUGGESTING DISLOCATION IN ADDITION TO FRACTURE OF HEAD.

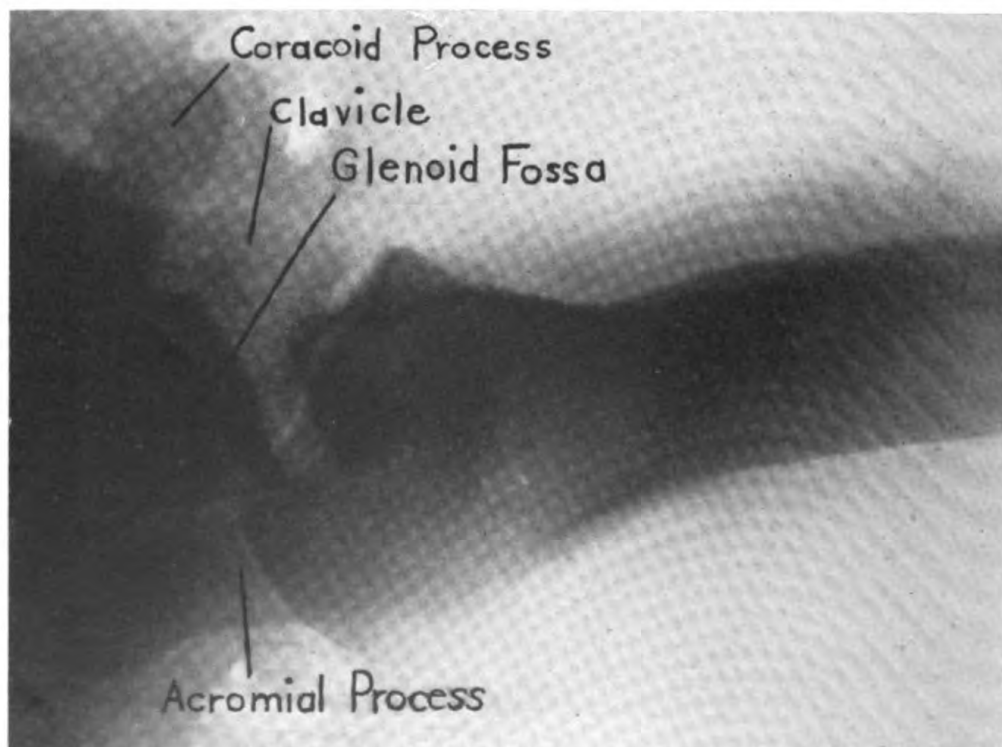


FIGURE 3B.—CASE 3.—VERTICAL VIEW SHOWS THE GASH FRACTURE OF THE HEAD IN PROFILE WEDGED ON THE POSTERIOR RIM OF THE GLENOID.

cases of shoulder injury in which the anterior-posterior view is not entirely satisfactory, when symptoms are not promptly relieved by treatment, and in all cases of fracture about the shoulder girdle, including the upper end of the humerus.

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VENEREAL DISEASE EDUCATION IN THE NAVY

A QUESTIONNAIRE STUDY WITH COMMENT AND SUGGESTION FOR IMPROVEMENT

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One year ago, the admission rate for venereal diseases aboard the vessel of which the author was the medical officer was higher than the usual expectancy. At the time, this vessel was making regular trips to the West Indies and return.

In an effort to reduce the venereal rate, the medical officer carefully questioned each new admission, and it was found that there was a definite lack of knowledge of the essential facts of the venereal diseases and their prophylaxis. Even fundamental facts of each disease were either unknown or badly distorted, and their methods of prophylaxis were definitely inadequate. This was an unpleasant discovery to the medical officer, who had regularly given carefully studied lectures on the subject to the entire crew, followed by the usual invitation to ask questions on anything that was not thoroughly understood. All had listened with apparent interest, but now it was obvious that the lectures had not gotten across to at least a part of the crew.

To determine fairly accurately the degree of practical knowledge of the venereal diseases and their prophylaxis that the complement of

the vessel had, a questionnaire was prepared, and with the helpful cooperation of the commanding officer, was given one afternoon at sea to the entire ship's company, from the commanding officer down.

It is believed noteworthy to state that the papers as a whole were carefully and thoughtfully written. There was little, if any, facetiousness.

THE QUESTIONNAIRE

(This questionnaire is to be answered by the entire ship's company. It will be used by the medical officer only, in compiling statistical data, after which all papers will be destroyed. In the last section, make your criticism frank and constructive. The papers will not be graded and marks will not be given. Names are to be written on the last page of your paper so that a check can be made that a paper has been received from every person aboard. If you do not know the answer to any particular part, please so state.)

1. State what you know concerning gonorrhea (clap) including how it is contracted from an infected person, how the germ enters the body, its first signs, and the after-effects of the disease.

2. State what you know concerning syphilis, including how it is contracted from an infected person, how the germ enters the body, its first signs, and the after-effects of the disease.

3. Fundamentally, how does gonorrhea differ from syphilis?

4. State what you know about prophylaxis (measures of prevention) after exposure to venereal infection, including in detail, what measures *you* take to prevent venereal disease after exposure.

5. How much naval service (years) have you had?

6. Approximately how many lectures on venereal diseases have you been given by medical officers?

7. Do you feel that you have received much worthwhile information from these lectures?

8. What is your criticism and suggestion for improvement on the subject of venereal-disease control in the Navy?

TABLE 1.—*Results of questionnaire for entire group*

S—A satisfactory knowledge. D—A distorted knowledge. O—No knowledge

	S	D	O
Question No. 1.....	125 (63%)	51 (25%)	24 (12%)
Question No. 2.....	67 (34%)	96 (48%)	37 (18%)
Question No. 3.....	95 (48%)	105 (51%)
Question No. 4.....	102 (51%)	78 (39%)	20 (10%)

Average lectures for group, 8.

TABLE 2.—*Group comparison*

Group No. 1.—Men having less than 1 year service (55)

S—A satisfactory knowledge. D—A distorted knowledge. O—No knowledge

	S	D	O
Question No. 1.....	27 (50%)	18 (32%)	10 (18%)
Question No. 2.....	11 (21%)	32 (57%)	12 (22%)
Question No. 3.....	14 (25%)	41 (75%)
Question No. 4.....	24 (44%)	26 (47%)	5 (9%)

Average lectures for group, 4.3.

Group No. 2.—Men having 1-4 years service (63)

S—A satisfactory knowledge. D—A distorted knowledge. O—No knowledge

	S	D	O
Question No. 1.....	44 (70%)	14 (22%)	5 (8%)
Question No. 2.....	23 (37%)	23 (44%)	12 (19%)
Question No. 3.....	27 (43%)	-----	36 (57%)
Question No. 4.....	34 (54%)	23 (37%)	6 (9%)

Average lectures for group, 6.

Group No. 3.—Men having history of venereal disease (66)

S—A satisfactory knowledge. D—A distorted knowledge. O—No knowledge

	S	D	O
Question No. 1.....	41 (62%)	14 (21%)	11 (17%)
Question No. 2.....	13 (19%)	33 (50%)	20 (31%)
Question No. 3.....	23 (38%)	-----	41 (62%)
Question No. 4.....	17 (26%)	36 (55%)	13 (19%)

Average lectures for group, 9.8.

COMMENT

The general lack of knowledge of the subject was greater than anticipated. The most striking point that the questionnaire brought out was the distortion of fact. A grain of truth would be mixed with a pound of hearsay fiction to form a false and sometimes dangerous conception. Many of the ideas expressed were of the type heard around the corner drug store. For example, one man who has had 9 months naval service and who professed to have received much benefit from four lectures by naval medical officers said, "You get clap from a girl who is having her sickness."

Other examples of the weird ideas on the subject were:

When you get syphilis, your brain is full of pus. If I got it, I would commit suicide.

Syphilis is another name for leprosy.

You can get syphilis by eating from dishes that someone who had syphilis used, even after they are washed.

Syphilis usually causes cancer later in life.

You can get syphilis from a drinking glass or towel. And you can get it from a girl, too.

I know a man who had a bone broken, and the Doc. didn't set it right, and he got syphilis.

Gonorrhea is a disease of the muscles.

You can get clap from a strain or from a toilet.

Gonorrhea is worse than syphilis, because it can't be cured.

If a man gets clap, all his children will be born blind.

These quotations are isolated to illustrate how facts on the subject were distorted. In some instances, aside from a quotation as noted above, the rest of the paper was fairly satisfactory. It should be

noted that these misconceptions were by no means confined to the men new to the service. For example, as can be seen by the charted results of the entire group, 34 percent of the entire group had a satisfactory knowledge of the primary facts of syphilis, while 66 percent had their facts distorted or had no facts at all.

The most alarming misconceptions of all were those regarding prophylaxis. A few examples:

Prophylaxis should be taken twenty-four hours after intercourse.

It is better to take a "pro." shot 2 hours after being with a girl than right away. A doctor told me so.

While the above quotations are not typical of the whole group, they illustrate a distortion of fact which was found in a very large group. Of the entire group, 51 percent had a satisfactory working knowledge of prophylactic measures, while 49 percent had this knowledge dangerously distorted or degenerated into a lot of useless hocus-pocus. The seriousness of this is obvious. And the average number of lectures from medical officers for the entire group was eight.

COMMENTS ON GROUP COMPARISON

In the group with less than 1 year of service, the scores on all questions were appreciably lower than those of the entire group or for the group with 1 to 4 years' service. This in spite of the fact that the average lectures given to this group on the venereal diseases was 4.3 in 9 months. Many in this group professed to having had much instruction on the subject in the National Guard or Reserve groups before entering the Navy.

The group having a history of venereal disease gave a definitely poorer score than that of the entire ship or that of the 1- to 4-year service group. Of the 66 men in this group, 76 percent still had a distorted or completely unsatisfactory method of prophylaxis. And the average lectures on the subject for this group was the highest of all groups—9.8.

Of the 200 men examined, 36 turned in papers which demonstrated that they had an entirely satisfactory knowledge of the subject. Of this group, only 4 had a history of venereal infection, or a ratio of 1 to 9. Of the 164 whose papers were either partly or entirely unsatisfactory, 62 had a history of venereal infection, or a ratio of 1 to 2.6.

It was a peculiar coincidence that 10 percent of each group reported that they did not believe they had received any benefit from the venereal lectures. Oddly enough, most of this 10 percent were very well informed.

THE ANSWERS TO QUESTION EIGHT

In response to the request for criticism and suggestions for improvement, a number of worth-while ideas were given. Some of the sug-

gestions, while undoubtedly good, were too far advanced for the present era, when the populace at large still abhors hearing even the names of the venereal diseases. A cross section of the answers to this question follows:

Have frequent dances for sailors to meet nice girls, so they won't have to pick them up in bars and on the streets.

Have movies instead of lectures on venereal disease.

Have organized athletic games for everyone so that everyone can play hard and use his energy that way.

Make it easier for the sailors to wear civilian clothes ashore, so that they will have equal opportunities with civilian boys in meeting nice girls. Nice girls don't like to be seen with sailors in uniform as they are too conspicuous.

Keep the prophylactic record sheet more private. After putting his name on this sheet he is subjected to a lot of kidding and gossip.

Have slips of paper and a locked box with a hole in it at the prophylactic station. Then when a man takes a shot he can write his name on a slip and drop it in the box.

Have government controlled prostitute houses.

The ship's doctor should pick out the best house and examine the girls and then put a S. P. outside and don't let anyone but our boys in it.

DISCUSSION

It is believed that the present system of disseminating venereal disease information in the Navy is not entirely satisfactory. A lecture on this subject must of necessity contain many concise related facts and our average audience is simply not able to grasp them all. As a result, a few facts are picked up which are coupled with the distorted knowledge the average adolescent boy gets from his "wise" buddies, and the resulting conception is unsatisfactory and sometimes worse than no knowledge whatever.

It is believed that a great mistake is being made in the common assumption that the average sailor is "wise" in things venereal. It is true that he is given a very liberal education along these lines by the circumstances of naval service. A sailor going ashore in a foreign port finds that he is barred by peculiar sociologic principles from the better homes, hotels, parties, etc., and he quite naturally, looking for fun, gravitates to the bars and dives where the sailor is made welcome. One has only to be aware of the venereal rate of ships visiting foreign ports—and certain domestic ones—to realize that the sailor is not able to cope with the situation. Conclusions drawn from the results of the questionnaire on preceding pages would indicate that his difficulty, at least in part, is due to ignorance.

The Navy has an exceptional opportunity to help in the national campaign on venereal disease education now in progress. This is particularly true at the present time when the Navy is being greatly expanded. Most authorities agree that definite progress will be made in conquering the venereal disease problem when the public becomes

sufficiently educated on the subject. If we, in the Navy, adopt a more definite venereal disease educational program, we should be able to lower our own venereal disease admission rate and at the same time render important assistance to the national campaign.

While the incidence of venereal disease in the Navy is probably not higher, when all factors are considered, than that of corresponding groups in civil life, the control of the personnel in the Navy offers a distinct advantage for educational purposes. Since the modern trend is toward education of the public on the subject of venereal disease in an effort to "bring it out in the open" and regard it more as an illness to be cured, rather than a disgrace at which to shake our heads, the time seems opportune for the Navy to take a more definite course in the education of its personnel on the subject.

We, in the Navy, have been far ahead of the country at large in the matter of education on this subject. We have for years been giving our men lectures, demonstrations, slides and movies; we have made available sanitubes and condoms; and we have established convenient prophylaxis stations ashore. We have taken definite action against the problem, and our measures have without doubt helped immensely in keeping our admission rate down. But we still have too many new venereal disease admissions each year. Conclusions drawn from the answers to our questionnaire would indicate that a certain percentage of these new admissions is due, either to no knowledge of the subject, or to false confidence in a distorted knowledge. It is with this group that it is felt progress can be made.

For an educational program on this subject to get across to our personnel, it should fulfill these conditions:

- (1) Is the material presented in a medium whereby it can be grasped, studied, and digested by the slow as well as the fast thinker?
- (2) Is the material presented in words and manner which the average enlisted man can grasp?
- (3) Is there any inducement for the average enlisted man to acquire this knowledge?
- (4) Is there a way of forcing the naturally sluggish group to acquire this knowledge?
- (5) Is the primary presentation of the subject matter supported by supplementary types of instruction?

Our present educational plan does not fulfill these conditions.

The author believes that the primary medium for an educational program on this subject should be the printed page. At first, a small printed instruction course covering all the essential facts should be prepared and presented to every man in the service and to all recruits as they come in. Later this material should be incorporated in the A-N course at its next printing. The material on this subject now in the A-N course is considered far too sketchy to be of any practical value.

The subject matter should be simply worded so that the average enlisted man can readily grasp it. It should be complete enough to cover all the essential points and yet not be so bulky or technical as to discourage him.

In the opinion of the author, knowledge of this material should be required along with the A-N course for all advancements in rating. It should be a requirement for all Annapolis classes, as well as for the recruit classes at the training stations. Requiring this material for all advancements in rating would provide the necessary incentive to study the subject and would thus overcome the natural sluggishness of some people to acquire knowledge unless it is forced upon them. This group, with a tendency to mental torpidity, is the one it is most important to reach.

It is recommended that supportive types of instruction such as discussion groups, motion pictures, and applied instruction be given. The discussion groups, utilizing talks, questions, and answers, would accomplish much more than the present lecture groups in that they would be only supplementing a knowledge which the audience already had. Motion pictures would be a useful supportive measure. These movies should differ from most of those now available which spend most of their footage showing the horrible results of late syphilis. They should include simple facts of each disease, with prophylaxis measures, done preferably by animated drawings. Further supportive instruction would be gained by having all venereal prophylaxis supervised by a competent corpsman, as is now being done on some ships. This would amount to practical applied instruction.

It is believed that all personnel who acquire a venereal disease should be retained on the ship's restricted list a sufficient period of time, after they are pronounced well, till they can pass a very comprehensive examination on the instruction course on the subject of the venereal diseases and their prophylaxis. It should be provided that if they do not pass such an examination completely satisfactorily, that they be made to wait at least 2 weeks before reexamination. This feature of the suggested program will force the needed knowledge on the group that needs it most. It is this group that does not have sufficient basic moral fiber, the "tough luck" group that did not know how to protect themselves in the past and, as our questionnaire indicates, do not take the trouble to obtain knowledge to protect themselves for future bouts.

It might be said that, by these recommendations, we are trying to make doctors or corpsmen of all our personnel. In answer to this, it should be remembered that we have been teaching first-aid for years. We require knowledge of it for advancement in rating, and page after page of our A-N course is devoted to the various "carrys," etc. Surely knowledge of the venereal diseases and how to guard

against them is "first-aid" knowledge. And this knowledge would be many times more useful and practical to either a peacetime or a wartime Navy than the usual first-aid knowledge—and it is not intended to belittle first-aid instruction. It must be obvious that the average enlisted man will never meet an emergency where the application of a tourniquet will be a life-saving measure, while this suggested course of instruction will cover our "Achilles' heel" in that it will protect our personnel in a situation that presents itself with every "liberty."

The criticism might be made that the suggestions offered here would only be encouraging our men to more venereal exposure than ever. Any intelligent person must know that education never weakened character. And since it is obvious that a certain group of our personnel will continue to expose themselves, there can be no good reason why we should not force a knowledge on them which might save them from undesirable consequences. The group with sufficient stamina to avoid all exposure will surely not be harmed by this instruction, but will be further entrenched in their good resolution.

There will be some to say that if we intensify our educational program on this subject, it will make our Navy too "venereal disease conscious." Others will say that such a plan would be in "poor taste." With 10,000 new venereal disease admissions each year, it would seem high time that the Navy became "venereal disease conscious." And the question of "poor taste" would disappear in the few months it would take to become accustomed to a slightly changed system. The subject knowledge should be an essential part of every man's education in this modern world, and if in dispensing this knowledge to our personnel, we offend certain sensitive souls, they will simply have to bear with us.

Another criticism that might be offered is that the results of the questionnaire used here are not indicative of the Navy as a whole, and whether a cross section of any single ship is sufficiently characteristic of the entire Navy to warrant conclusions being drawn. The point is debatable, of course, but certain statistics and past experience leads one to believe that it is true in this particular instance.

It is believed that if we are to make further progress in lowering our admission rate for venereal disease, it must be done along a more intensive educational line. The above suggested plan, or some better plan along this line, would seem to be well worth a trial.

What was thought to be a concise and informative presentation of the subject was prepared by the medical officer, and issued as an "educational course" to every man aboard the ship. It was well received and stimulated much discussion in the wardroom and crew compartments. Later, various groups were questioned and it was found that knowledge of the subject had materially improved. It is

believed worthy of mention to state that for the 6-month period following the issuance of this educational course, the incidence of venereal disease aboard this vessel was reduced by 50 percent over the 6 months period preceding it.

**TREATMENT REQUIREMENTS OF EARLY SYPHILIS AFTER NEGATIVE
SEROLOGY IS ESTABLISHED**

By Lieutenant Gordon H. Ekblad, Medical Corps, United States Navy

This paper, based on 1,000 cases of early syphilis followed from 3 to 20 years, attempts to arrive at some definite conclusion as to the optimum number of injections necessary after negative serology has been attained.

The data have been obtained from abstracts of the histories of over 1,500 syphilitic naval personnel, and were compiled during the period from 1936 to 1940. Syphilitic records were examined from naval bases, hospitals, battleships, cruisers, gunboats, destroyers, submarines, and miscellaneous ships. This is over 10 percent of the total syphilitics in the navy (prior to recent naval expansion) and should give an accurate index of the result of naval antisymphilitic treatment. From these fifteen hundred records, all cases meeting the following requirements were included for study:

(a) Three or more years since diagnosis and institution of treatment.

(b) Two or more years since completion of original treatment course.

(c) Only cases diagnosed as early syphilis are included,—i, e., seronegative primary, sero-positive primary, and secondary syphilis.

(d) Since standardization of treatment for only the normally responding cases is being attempted, cases remaining Kahn fast for more than 1 year after institution of treatment, and a few cases relapsing while actually under active treatment, were not included. It is felt that these cases require special consideration and cannot be standardized as regards the necessary treatment.

(e) Reinfections were not included for the reason that the criteria for reinfections are at extreme variance; by using one set of standards nearly all recorded reinfections would be judged as reinfections, or by using another set of standards nearly all recorded reinfections would be judged as relapses.

A total of 1,000 cases meeting the above criteria is presented as the data for determining an optimum amount of treatment after negative serology. During the past 6 years most syphilitics in naval personnel have received treatment under a continuous treatment schedule, and

a study of the results in another year or two will give a better comparison of cases on a continuous treatment regime than is true of the present series.

Of these 1,000 cases, 238 were in the 3- to 5-year group (3 to 5 years since diagnosis and institution of treatment); 405 in the 5- to 10-year group; 357 in the 10- to 20-year group. Seven hundred seventy-four of the cases were in the seropositive group, and 226 cases in the seronegative primary group; the 2 groups are considered separately.

RESULTS IN SERO-POSITIVE PRIMARY AND SECONDARY CASES

Table 1 gives a comparison of results to be expected, in early seropositive syphilis, from varying amounts of treatment received after negative serology has been obtained.

TABLE 1.—774 cases sero-positive primary or secondary syphilis.—Percentage of cases showing blood serology relapse after having once become negative

[3 to 20 years since diagnosis and institution of treatment; 2 or more years since completion of original treatment. Post-negative serology treatment = treatment received during the first year of negative serology, plus any treatment received during the second year of negative serology which is definitely a continuation of a previous treatment scheme. For complete data see table 3 (a)]

	Number of arsenical injections administered after negative serology						
	0	5	10	15	20	25	30
	Percent	Percent	Percent	Percent	Percent	Percent	Percent
Continuous treatment.....	50	50	8	9	5	0	4
Intermittent treatment.....	67	73	37	17	4	7	10
Inadequate treatment.....	72	41	51	11	33	20

From table 1: (1) Less than 20 injections of arsenicals after a negative test is not sufficient to prevent relapses.

(2) Apparently, with the continuous and intermittent treatment groups, not much can be accomplished by giving more than 20 arsenical injections after negative serology. Only 2 patients in the continuous or intermittent treatment groups received more than 30 arsenical injections after negativity, so that results of 40 or 50 arsenicals after negativity cannot be judged. However, this tremendous amount of treatment would probably do the patient's health more harm than it would benefit him in regard to his syphilis. This is particularly true in view of the fact that every one of the 6 patients in this series who relapsed after having received continuous treatment and 5 or more arsenical injections after negativity, and every one of the 40 patients who relapsed after having received intermittent treatment and 10 or more arsenical injections after negative tests, showed nothing more serious in their records than blood serology relapses, all of which were easily amenable to further routine treatment. Only 30 of these

patients had had spinal fluid examinations done after relapse—of the spinal fluids examined none showed anything more than a cell count elevation up to 11.

(3) None of the inadequate treatment cases received more than 25 arsenical injections after a negative serology. This is not sufficient treatment to prevent relapses in this originally inadequate treatment group. The data in this material does not indicate the proper amount of treatment necessary in these particular cases.

A study of the above data would seem to indicate that slightly less treatment is necessary in most patients than is usually advocated today; and that slightly more treatment is necessary in some patients than is usually given. The usual criteria advocated by different authorities varies between 14 to 18 months of continuous treatment in seropositive primary or early secondary cases.

Assume the following hypothetical case in which the patient becomes seronegative in 3 months. If 20 additional arsenical injections are sufficient, this patient would require 44 more weeks of treatment (for the sake of clarity, no account is taken here of the time difference which overlapping of bismuth and arsenical courses would make; 4 additional weeks of treatment are added to account for a longer bismuth course in ending treatment). This makes 13 weeks' treatment while seropositive, and 44 weeks' treatment after a negative test, or a total of 57 weeks' or 13 months' treatment. Similarly, a patient becoming seronegative in 6 months would require 16 months' treatment.

In the Navy, 63 percent of early seropositive cases receiving continuous treatment will be seronegative in 3 months, and another 28 percent of the original total will be seronegative in 6 months.¹ Based on the present findings these cases would then require 13 and 16 months' treatment respectively. The 9 percent of cases which received continuous treatment but required longer than 6 months to become seronegative will of course require a correspondingly longer time for treatment.

While the above schedule constitutes slightly less treatment for a large percentage of patients receiving continuous treatment than is usually advocated, note carefully from table 3 (a) that this is actually *more* treatment than 76 percent of patients in this series received after negative serology.

SERO-NEGATIVE PRIMARY SYPHILIS

Results of treatment of sero-negative primary syphilis in the Navy is shown in table 2.

¹ Ekblad., G. H.: U. S. Nav. Med. Bull., April 1939.

TABLE 2.—226 cases sero-negative primary syphilis—percent of cases showing blood serology relapse

[3 to 20 years since diagnosis and institution of treatment; 2 years or more since completion of original treatment. For complete data see table 3 (b) and 3 (d).]

	Number of arsenical injections					
	5	10	15	20	25	30
Continuous treatment.....	Percent	Percent	Percent	Percent	Percent	Percent
Intermittent treatment.....				0	0	0
Inadequate treatment.....	56	54	36	18	14	0

Sero-negative primary cases receiving continuous treatment showed no serologic relapses provided they received 20 or more injections of arsenicals. It would appear from table 2 that 25 injections of arsenicals are a safe amount of treatment. This corresponds roughly with the amount of treatment usually recommended for sero-negative primary cases of syphilis.

Intermittent treatment cases will require a minimum of 30 injections of arsenicals.

NEED FOR PHYSICAL AND SPINAL FLUID EXAMINATIONS

In closing, it is emphasized that the present standardization scheme is not intended in any way or means to replace careful physical and spinal fluid examinations, but to supplement them.

SUMMARY OF 1,000 CASES OF SERO-NEGATIVE PRIMARY, SERO-POSITIVE PRIMARY, AND EARLY SECONDARY CASES OF SYPHILIS

TABLE 3 (a).—Sero-positive cases, showing the number of cases receiving the stated amounts of treatment after negative serology had been attained

[3 to 20 years since diagnosis and institution of treatment; 2 or more years since completion of original treatment]

	Number of arsenical injections received after negative serology had been obtained						
	0	5	10	15	20	25	30
Continuous treatment.....	4	2	11	10	19	27	21
Intermittent treatment.....	23	7	38	59	65	25	9
Inadequate treatment.....	49	28	38	24	4	4	0

Figures in the boxes show the number of patients who had no blood relapses after having once become sero-negative.

	Number of arsenical injections received after negative serology had been attained						
	0	5	10	15	20	25	30
Continuous treatment.....	4	2	1	1	1	0	1
Intermittent treatment.....	47	19	22	12	3	2	1
Inadequate treatment.....	124	22	39	3	2	1	0

Figures in the boxes show the number of patients who had blood relapses after having once become sero-negative.

TABLE 3 (b).—*Sero-negative primary cases, showing the number of cases receiving the stated amounts of treatment*

	Number of arsenical injections received					
	5	10	15	20	25	30
Continuous treatment.....				6	15	28
Intermittent treatment.....				27	38	20
Inadequate treatment.....	7	13	23			

Figures in the boxes show the number of patients who had no blood relapses.

	Number of arsenical injections received					
	5	10	15	20	25	30
Continuous treatment.....						
Intermittent treatment.....				6	6	
Inadequate treatment.....	9	15	13			

Figures in the boxes show the number of patients who had blood relapses.

TABLE 3 (c).—*Results of different treatment schemes in early sero-positive syphilis*

	Cases	Percent blood relapses
Continuous treatment.....	104	10
Intermittent treatment.....	332	32
Inadequate treatment.....	338	57
Total.....	774	40

TABLE 3 (d).—*Results of different treatment schemes in sero-negative primary syphilis*

	Cases	Percent blood relapses
Continuous treatment.....	49	0
Intermittent treatment.....	97	12
Inadequate treatment.....	80	46
Total.....	226	22

Kahn-fast cases of 1 year or more, and reinfections, are not included in this series of cases.

Continuous treatment.—Weekly treatment of an arsenical or heavy metal; no treatment lapse during the first 6 months. Cases with lapse of 1 month in treatment during the second 6 months are included in this group. A minimum of 20 arsenical injections during the first year of treatment.

Intermittent treatment.—Twenty or more injections of arsenicals, plus an equal number of bismuth injections, during the first year of treatment, via any schedule.

Inadequate treatment.—Less than 20 injections of arsenicals in the first year of treatment.

Arsenicals.—The type of arsenical used in all but a small number of these cases was either neo-arsphenamine or mapharsen.

Bismuth.—Practically all of these cases received at least as many bismuth injections as arsenical injections. Some of these patients, particularly in the older groups, received mercury instead of bismuth, or a combination of mercury and mixed treatment, or a combination of mercury and mixed treatment and bismuth. No attempt was made to segregate these various combinations.

Post-negative serology treatment.—Treatment received during the first year of negative serology, plus any treatment given during the second year of negative serology which is definitely a continuation of a previous treatment scheme.

SUMMARY

1. Data is submitted indicating that the following standardization of treatment may be adequate for normally responding cases of early syphilis:

(a) Seronegative primary cases (with normal physical and spinal fluid findings):

Continuous treatment:

25 arsenical injections.

25 bismuth injections.

Intermittent treatment:

30 to 35 arsenical injections.

30 to 35 bismuth injections.

(b) Seropositive primary and early secondary cases (continuous or intermittent treatment):

Twenty arsenical injections, and 20 bismuth injections, after negative blood and spinal fluid serology.

2. In early seropositive cases becoming negative in less than 6 months, the above recommendation represents a slight reduction in the amount of treatment from that usually accepted as adequate. In early seropositive cases becoming negative in more than 6 months, the above recommendation represents a slight increase in the amount of treatment from that usually accepted as adequate.

3. Only 24 percent of the cases in this series received as much treatment as the above recommendation calls for.

4. This standardization is based upon actual findings and is submitted for consideration as it represents a simple yardstick for measuring the amount of treatment still required in early seropositive syphilis after negative serology has been obtained.

CLINICAL NOTES

KERATOCONUS FOLLOWING CORNEAL TRANSPLANT

By Lieutenant Commander C. A. Swanson, Medical Corps, United States Navy

The case to be reported is believed to be the first recorded instance of keratoconus as a sequel to a corneal transplant.

Keratoconus involves a stretching of the cornea in its axial part resulting in visual impairment; it is a noninflammatory ectasia, or in a sense an anterior myopia.

The condition was first noted by Mauchert in 1748 (1), and first adequately described by Nottingham in 1854 (2). The cause of the condition is not known, although there has been much theorizing that the condition has been due to endocrine dyscrasia with hypothyroidism being held the glandular deficiency most likely responsible, as noted in articles by Knapp (3) and Sitchevoka (4).

Keratoconus is almost invariably bilateral and as a rule manifests itself about the age of puberty. The ectatic portion is usually confined to the central portion of the cornea and the apex of the curve being eccentrically placed downward and nasally. The normal height of the cornea is about 2.5 mm. and the protrusion of the apex rarely amounts to an additional 2 mm. Rupture is unknown.

In the early stages the cone is difficult to see. The easiest way to observe it and note the character of its outline is when standing in front of the patient and raising the upper lids, to ask him to look down so that the free margin of the lower lid bisects the cornea horizontally so that the angular curve assumed by the lid margin is a measure of the conicity and is easily apparent and is called Munson's sign (5).

The symptom complained of is a gradual visual deterioration. Ordinary lenses cannot compensate for the hyperbolic curve and are of little value. Recourse now may be had to the contact glass with good visual result.

CASE REPORT

The following case is of interest for medical record. Prior to admission to hospital vision in each eye had always been recorded as 20/20 on annual physical examination. The patient (P. N., Jr.) was first admitted to the United States Naval Hospital, Pearl Harbor, T. H., November 20, 1939, with corneal ulcer of the right eye. The eye was quite painful and accompanied by marked lacrimation and blepharospasm. There was a branching infiltration of the cornea which had

the clinical appearance of dendritic keratitis. The physical examination was negative excepting a 20-percent loss of hearing in right ear. Typhoid vaccine was given without result. This was followed by Vitamin "A" medication and sulfanilamide without apparent improvement. Ultra violet light was next tried and the ulcer began to heal. In January 1940 the thermophore was applied locally and on February 10, 1940, the ulcer was healed with a leucomatous scar involving $\frac{3}{8}$ the area of the cornea. Vision OD 20/200; vision OS 20/20.

In May 1940 the patient was seen in consultation by Dr. Ramon Castroviejo of New York City, who recommended corneal transplant. The patient was placed on riboflavin and was admitted to the Eye Institute, New York City, July 1, 1940, with a diagnosis of corneal opacity right eye. July 3, 1940, a keratoplasty was performed by Dr. Castroviejo. Post-operative eye reaction was good, the sutures being removed July 8, 1940, and on July 13, 1940, a note was made that there was no displacement of the transplant and that the anterior chamber was of normal depth and there was no corneal edema.

On July 27, 1940, the patient took a trip to Washington and vision in the OD was 16/20 or .8 vision on the Clason visual acuity meter. The corneal transplant was clear and the fundus appeared normal. Several days later, on August 1, 1940, atropine was ordered instilled in the right eye in New York. A cyclitis developed and typhoid vaccine was given. The patient was discharged to duty under observation August 27, 1940.

On September 27, 1940, the corneal graft appeared quite clear except for pigmented keratitic precipitates on Descemet's membrane which precluded clear vision. Vision OD 20/200 (2/20). Dionine was prescribed. A few cells could be seen in the aqueous by slit lamp examination. The fundus was not very clear because of the heavy keratitic precipitates.

In February 1941 the keratitic precipitates had wholly cleared and no cells could be seen in the aqueous. With the ophthalmoscope an annular dark shadow separated the bright red reflex of the central and peripheral areas. The fundus was too hazy to be studied in detail. Vision OD 20/200.

In April 1941 Munson's sign was positive. There was anaesthesia of the transplant. On slit lamp examination there was noted a definite thinning of the corneal cone at the apex. An endothelial reflex appeared in the central portion of the cornea at the peak of the cone due to increased concavity of the posterior surface. No nerve fibers could be seen in the transplant. The patient was examined with a contact lens and with a 2/8 (12 mm. scleral radius and 8 mm. corneal radius) contact lens vision in the OD was 20/20. The fundus was perfectly normal. There was a pigment precipitate on the anterior lens capsule not in the visual axis. The lens was clear.

Search of the literature failed to reveal keratoconus as a complication of keratoplasty.

Photographs of the patient's eyes show well the left eye with a normal corneal curvature, and the right eye with a conical corneal curvature. See Figures 1 and 2.

Plaster of Paris casts of each eye were made from Negocoll impressions and photographed. Figures 3 and 4 show the casts as the corneae would appear as viewed from below upward. The corneae are well outlined in this figure by a pencil line. One can clearly see the apex of the cone of the right eye displaced nasally. Although not shown, the apex of the cone is displaced downward in the right eye. Actually, then, the apex of the cone is situated eccentrically downward and nasalward.

Measurements were next taken on a Bausch and Lomb Keratometer, and the normal left cornea was found to have a corneal curvature of $42\frac{1}{2}$ diopters (42.50) equal to a 7.94-mm. corneal radius of curvature. No reading could be obtained on

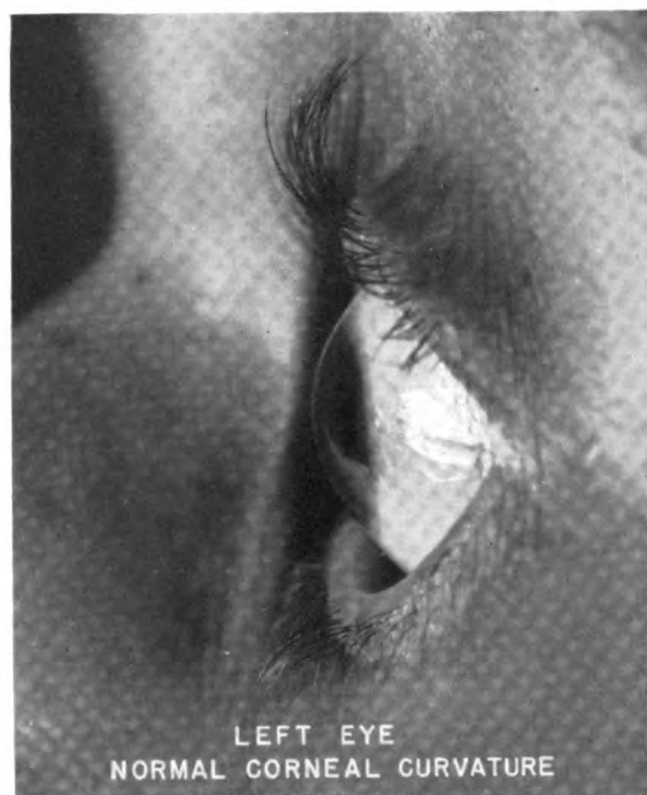


FIGURE 1.—LEFT EYE. NORMAL CORNEAL CURVATURE.

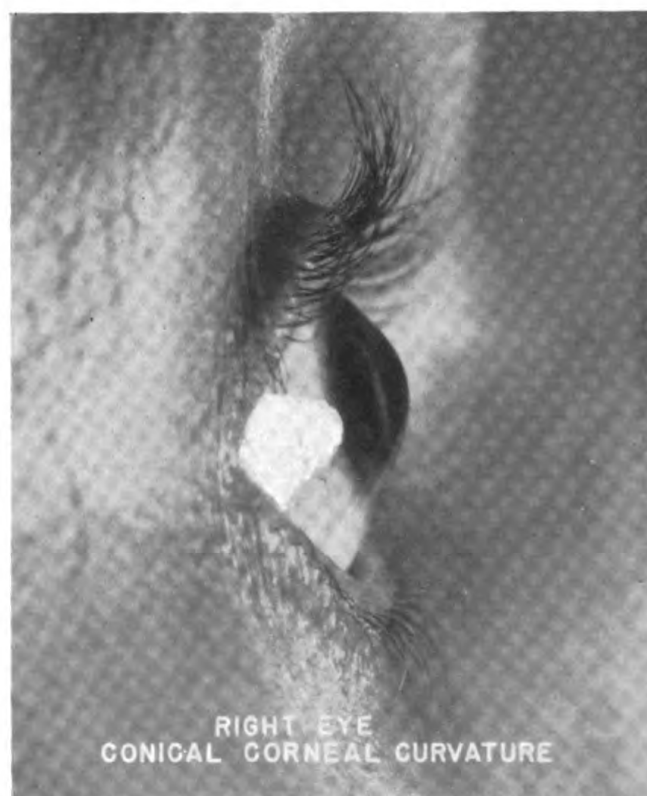


FIGURE 2.—RIGHT EYE. CONICAL CORNEAL CURVATURE.

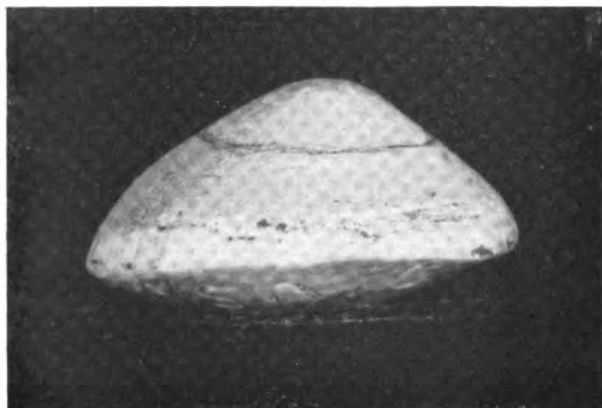


FIGURE 3.—RIGHT EYE CAST.



FIGURE 4.—LEFT EYE CAST.

the right cornea, and even at 52 diopters (52) of corneal curvature (6.75 mm. radius of curvature) the corneal images were indistinct. The normal average corneal curve is 44 diopters. Irrefutable evidence has been presented of this being a case of keratoconus. The patient is now being fitted with a plastic contact lens.

CONCLUSION

1. A case of Keratoconus is presented and believed to be the first such case recorded as a sequel to corneal transplant.

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ANEURYSM OF AXILLARY ARTERY WITH CEREBRAL EMBOLUS

A CASE REPORT

By Lieutenant Commander Gerald W. Smith, Medical Corps, United States Navy

This case is of unusual interest because of the diagnostic problem presented. In a review of the literature no case report could be found of an axillary aneurysm which had developed a spontaneous thrombus which separated and produced a cerebral embolus.

L. P., seaman first class, United States Navy, aged 22 years, was admitted to the naval hospital February 17, 1939, from a naval vessel.

The preceding night he had reported to the sick bay on board ship complaining of slight dizziness. He awakened early the next morning and requested a shipmate in the next bunk to help him turn over because his left arm and leg were weak. He had voided involuntarily during the night. Within half an hour he was semistuporous.

History.—His past history was essentially negative except that he had complained occasionally of a tingling sensation in his right arm and hand. This symptom was never severe enough to cause him to report to the medical officer aboard ship for treatment.

Examination.—Physical examination on admission to hospital revealed a well-developed, muscular young man in an unconscious condition. Temperature by rectum was 99.2 F., respiration rate was 15, and pulse rate 80 per minute. The blood pressure was 120 systolic and 80 diastolic, taken on the left arm. Heart and lungs were negative. The right arm was somewhat cyanotic and colder than the paralyzed left arm. It was noted that the pulse could not be obtained on the right. Both common carotid arteries were pulsating normally.

The neurological examination showed the right pupil to be 6 mm. and the left pupil 2 mm. in diameter. Both pupils reacted sluggishly to light stimulation. There was a weakness of the left external rectus muscle causing the eye to deviate

nasally. The face was asymmetrical and showed a weakness of the lower two-thirds on the left side. The left upper and lower extremities were flaccid and completely paralyzed. The ulnar, radial, biceps, triceps, patellar, and ankle reflexes were exaggerated on the left. The left abdominal and cremasteric superficial reflexes were abolished though present on the right. Babinski group of signs were positive on the left with a positive left Hoffman sign. Painful stimulation on his left extremities produced no response. The left corneal reflex was abolished though it was present on the right. Skin on the paralyzed side showed less moisture than on the other side.

Laboratory results.—The spinal fluid was under an increased pressure of 32 mm. of mercury, clear in color, contained 5 cells per cu. mm. and its globulin content was not increased. Blood and spinal fluid Kahn tests were negative. Red blood cells were 5,500,000 and white blood cells 15,900 per cu. mm. with 72 percent polymorphonuclear cells, 10 percent lymphocytes, 16 percent eosinophiles and 2 percent mononuclears. Urinalysis was negative.

Clinical course.—The coma gradually deepened with abolition of the corneal and all deep reflexes. The blood pressure rose to 150 systolic and the diastolic pressure dropped to 40 giving a pulse pressure of 110. Cheyne-Stokes respiration developed and the temperature rose to 106° F. Death ensued 4 hours following admission to the hospital.

Autopsy report.—A sacculated aneurysm of the proximal part of the right axillary artery, size of a large olive, was found, which extended anteriorly and inferiorly and pressed against the brachial plexus (fig. 1). A thrombus filled the aneurysm and extended for 5 cms. distally, completely occluding the axillary artery. The thrombus also extended proximally occluding the right subclavian and innominate arteries, stopping at the point where the right common carotid leaves the innominate artery. A thrombotic embolus occluded the right internal carotid artery in the carotid canal of the skull. The right cerebral hemisphere was edematous and softened, especially in its middle and anterior portions. Small petechial hemorrhages were scattered throughout the cortex and small sub-arachnoid hemorrhages were present over the frontal parietal region of the right cerebrum.

Comment.—The thrombus had evidently extended proximally from the axillary artery to the junction of the innominate and right common carotid arteries. The current of blood surging upward and through the common carotid artery had separated a portion of the thrombus forming an embolus. This embolus had been forced upward along the course of the common and internal carotid arteries where it had become lodged in its carotid canal portion at the base of the skull. An anoxia of the right cerebral hemisphere developed. As the collateral circulation, mainly from the vertebral arteries, had been insufficient, cerebral edema and softening of the right cerebral hemisphere, especially in its anterior and middle portions occurred. Symptoms of left hemiplegia, coma and Cheyne-Stokes type of respiration ensued with resultant death of the patient.

The blood and spinal fluid serological examinations were negative for syphilis. There was no history or evidence of any trauma to the axillary region. Autopsy failed to reveal the presence of an infectious or degenerative process.

The etiological factor of this axillary aneurysm is considered congenital. The thrombus probably developed as a result of circulatory stasis within the walls of the aneurysm. The fact that the onset of the patient's symptoms occurred while he was sleeping adds weight to this assumption.



FIGURE 1.—SACCULATED ANEURYSM OF RIGHT AXILLARY ARTERY AFTER SECTION. INSERT SHOWS EXTENSION OF THROMBUS INTO RIGHT SUBCLAVIAN ARTERY.

PNEUMOCEPHALUS

CASE REPORT

By Lieutenant Commander Charles F. Flower, Medical Corps, United States Navy

On March 31, *T. H. P. F. 2c.*, Age 20, was a passenger in an automobile that collided with a truck. He was rendered unconscious and was apparently seriously injured.

Following a brief period at the Los Angeles Emergency Hospital he was admitted aboard the *U. S. S. Relief*, where his injuries were found to consist of a compound fracture of the skull involving principally the frontal bone on the left side, and a compound fracture of the left tibia and fibula.

He remained in a semicomatose condition for about 48 hours, had a blood tinged cerebral fluid draining from the left ear for 8 days, a marked left exophthalmos, and strabismus with resultant diplopia. Unilateral vision was good, and no signs of increased intracranial pressure developed, doubtless due to the decompressing effect of loss of cerebral fluid from the left ear.

On April 13, he was transferred to the Mare Island Naval Hospital. His chief complaints were headache, left earache, and double vision. The exophthalmos was still present, the otorrhea had stopped, and the fractures of the leg were reduced and immobilized in a cast.

A few days after admission the patient stated that he occasionally felt fluid run down into his throat from his nose, and, after 10 days, when he was allowed to sit up in bed, noticed a clear fluid run from his left nostril. This rhinorrhea continued off and on until early in August.

The exophthalmos subsided in 2 weeks, but the diplopia was present until June 1.

Radiographic examination of the skull on April 16, was reported as follows: "There is a hole in the frontal bone, measuring about 5 mm. in diameter, situated about 4 cm. above the left orbit. Fracture lines involving both inner and outer tables are seen radiating upward in the frontal bone from this point for a distance of about 4 cm. Two fracture lines radiate downward, one involving the inner table and the other the outer table of the frontal sinus. The line involving the outer table stops at the supraorbital ridge. The line involving the inner table goes into the orbit, crosses the sphenoidal fissure, and enters the left maxillary antrum. There is a large collection of air in the left frontal region which is apparently compressing the frontal lobe." (Fig. 1.)

With the evidence of rhinorrhea, and the radiographic demonstration of air in frontal region, pneumocephalus was diagnosed.

The patient's condition steadily improved, the fractured leg united, the diplopia and exophthalmos disappeared, he became ambulatory and led a perfectly normal life, except for the occasional rhinorrhea, which finally stopped. Thinking that he had obtained a spontaneous closure of the dura and thus recovered from the condition resulting in pneumocephalus, he returned to duty on September 19.

On November 10, he was readmitted by transfer from his ship, in a comatose condition with a history of having been found in his bunk having a convulsion.

Examination on his second admission showed him to have a flushed face, hyperpyrexia, and a bloody spinal fluid.

In a few hours he became semiconscious though stuporous, but the following day again became comatose, and had a series of 17 convulsive seizures. A day later he was alert, fully conscious, free from fever, and his conduct, speech, actions were entirely normal, and had no subjective complaints.

The rhinorrhea developed again 2 days after admission and continued for 4 weeks. As much as 130 cc. of fluid was collected in a 24-hour period. Radio-

graphic examination of skull on November 19, disclosed an irregularly outlined pneumocele in left frontal area, and air in the ventricle.

The patient remained symptom free for a month, when he had 3 days of distress characterized by nausea, vomiting, some increase in temperature (101°) and neck stiffness.

He again felt well until December 25, when, following a mild upper respiratory infection, he developed a left otitis media. Paracentesis was performed, and drainage continued for a month.

January 14.—The patient, who had been feeling very comfortable and whose middle-ear disease was rapidly improving began to experience generalized headache. His temperature mounted rapidly to 104° , accompanied by increased pulse rate 120 to 140 and respirations of 30 to 35. He had more convulsive seizures and became comatose. Avertin by rectum controlled the seizures. Spinal puncture at this time showed the fluid to be blood tinged. He rapidly recovered from this attack, appeared and felt very well in a few days.

February 8.—Developed a fever of 101.4° accompanied by right-sided headache, nausea, and vomiting.

February 9.—The temperature mounted to 101.8° with pulse rise to 104. The headache was more severe and a cerebral fluid rhinorrhea was present.

February 10.—There was further rise in temperature to 103° with gradually increased pulse rate 120 to 150. Restlessness, headache severe, pain in posterior cervical region.

At 6:55 p. m. had convulsive seizure, followed closely by several others, patient comatose. Death occurred at 8:10 p. m.

AUTOPSY REPORT

Cranium opened: Linear fracture, about 10 cm., seen in left frontal region extending to sphenoid bone. In the left orbital plate this fracture is approximately 5 mm. wide and 15 mm. in length. This opening is occluded by inspissated pus and fibrin. Probe easily passed from within the cranial cavity to the upper side of eyeball protruding beneath the supraorbital foramen. From the lower part of fracture, probe, if directed medially extends into left frontal sinus and on into the nose. This is apparently the route of the antemortem rhinorrhea. The dura in the region of this fracture is adherent to the brain. It is pierced by a hole 2 mm. in diameter above the fracture.

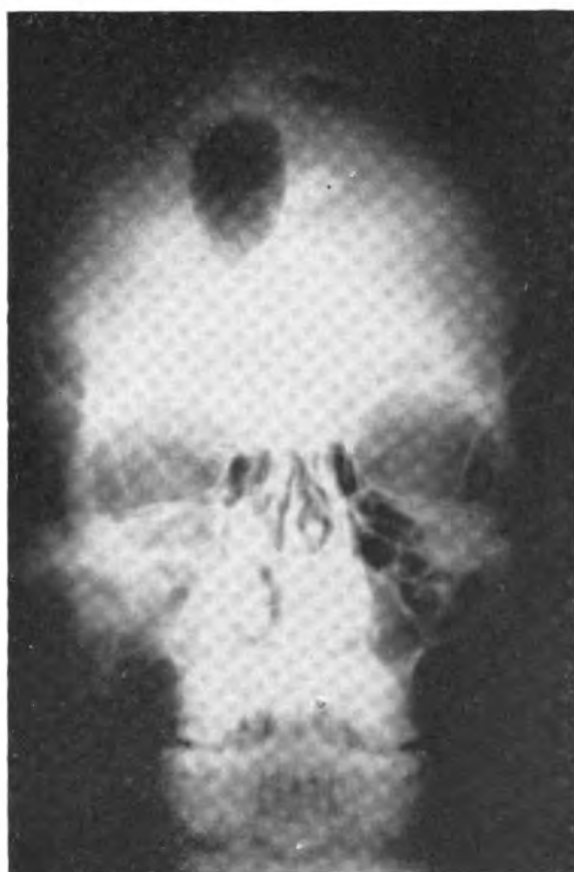
There is a cavity 5 cm. in diameter in the medial part of the left frontal lobe. This is seen by transillumination to contain air and straw colored fluid. This cavity communicates with anterior horn of the lateral ventricle. There is no gross pus in the brain or on the parts of meninges. The anterior horn of right lateral ventricle is greatly expanded.

The cerebrospinal fluid seems to have been under pressure and is somewhat blood tinged.

The entire cerebrum is congested, each small blood vessel being engorged, giving the brain a reddish appearance. There are petechial hemorrhages under the pia on the left side and at the base of the brain.

COMMENT

While perhaps not rare, pneumocephalus, following skull fracture, can be considered uncommon and report of a case is believed to be indicated in order to keep in mind this possible complication of our head injuries.



DORSO-VENTRAL VIEW OF SKULL SHOWING PNEUMOCELE IN LEFT
FRONTAL AREA.

Question will naturally arise, what is to be done for these people. Operation for repair of the torn dura is indicated where rent is favorably located. We thought the tear was multiple and in a region said by some authorities to be unfavorable for approach (viz: the region of sphenoid or anterior ethmoid sinuses).

Autopsy indicated the site of tear to be approachable, and it is reasonable to suppose repair might have been possible.

Our release of the man to a duty status on first admission was predicated on the supposition that spontaneous closure of the dura had been effected.

OTITIC MENINGITIS WITH DUAL INFECTION OF SPINAL FLUID

REPORT OF CASE

By Lieutenant Commander Roy W. Key, Medical Corps, United States Naval Reserve

Double infection of meninges and spinal fluid is uncommon. Tripoli (1) reported 422 cases of meningitis and found only two cases of double infection, both of which were fatal. In one of these cases, *Haemophilus Influenza* (Pfeiffer's Bacillus) was isolated once with staphylococci and once with pneumococci, respectively. In this same series there were 220 cases of meningococcic meningitis, with fatality rate of 65 per centum. There were 22 cases of streptococcic meningitis, with a fatality rate of 92 per centum. Cadham (2) states that in streptococcic meningitis the death rate was 97 per centum, and quotes H. Gray, who was able to find only 66 cases of record of recovery from streptococcic meningitis and six of these were in cases where the patient had suffered from scarlet fever. Pipirs (3) in a comprehensive article reported a study of 46 cases of mixed meningeal infections in which the tubercle bacillus predominated as the primary organisms, with the patient later to be infected with varied organisms, meningococci, pneumococci, and influenza bacilli predominating. These 46 cases had been reported by the German, French, English, and American writers and had extended over an interval from 1896 to 1927. Pipir's conclusions were that mixed meningeal infections were, by no means, such a rarity, but that recovery was indeed rare regardless of what treatment was used. Ashmun (4) reports five cases of pneumococcic meningitis combined with streptococcus and diplococcus catarrhalis. Three of Ashmun's five cases recovered, and all received optochin hydrochloride intraspinally, and neutral acriflavin intravenously.

Arnett (5) reported a case of meningococcic and staphylococcic meningitis not otitic in origin, in which the patient recovered. In Arnett's case the staphylococcus did not appear until the eighteenth day, and only after several lumbar and cisternal punctures had been done. Cunningham (6) in January 1937 reported a case of dual infection of the spinal fluid, in which there was first meningococci to

be followed later by appearance of hemolytic streptococci. His patient had as the onset follicular tonsillitis and left otitis media, in which a left mastoidectomy was performed, during which the dura was incised and in the spinal fluid thus obtained was isolated gram negative diplococci which was later diagnosed as meningococci. Three days later fluid from the right ventricle gave a pure growth of hemolytic streptococci, while fluid from the cistern gave a growth of meningococci. Cunningham was of the opinion that antistreptococcic serum was of little or no value in these cases and stated also that in meningococcic infections there should be a spinal drainage with intensive serum treatment both intravenously and intrathecally. He stressed further that in cases of mixed infections there should be active antimeningococcus therapy combined with immediate operation on the infected mastoid, but unless there was clinical evidence of brain abscess the dura should not be opened.

The case which we wish to present occurred in a young girl 13 years of age, who was moderately well nourished, but was subject to recurrent attacks of pharyngitis and laryngitis. She was seen by me on February 27, 1937, complaining of pain in the left ear. At that time there was only slight bulging of the ear drum, with a moderate elevation of temperature, and it was decided to use local treatment instead of myringotomy. However, the patient's pain and discomfort increased, as well as the temperature, and on March 1 the left ear drum was opened under ethyl chloride anaesthesia. There was a profuse drainage of a serosanguineous fluid which gave the patient some relief from the aural pain and there was apparent subsidence of mastoid tenderness. However, 9 days following, the patient's temperature was 103° (F.) and she became acutely tender over the left mastoid and was admitted to the hospital and advised to undergo a simple mastoidectomy, which operation was carried out on the day of admission.

Upon opening the cortex a very extensively diseased mastoid was found, and the surgical pathology was of such nature to cause us to make a tentative diagnosis of streptococcic mastoiditis, and with this in view, Prontosil was administered while the patient was still on the table, and after recovery from the anaesthesia, Prontylin was instituted by mouth. The mastoid infection was so extensive and was of such a nature that the removal of all cells and necrotic bone necessitated an uncovering of a small portion of dura in the middle fossa, as well as an area about $\frac{1}{2}$ by $1\frac{1}{2}$ cm. of the lateral sinus. In view of the surgical pathology found it was decided to leave the mastoid cavity open to better facilitate observation of the wound and to promote drainage. The patient's temperature fell by lysis and became normal on the fourth day following the operation. The patient was comfortable and there was a moderate drainage from the antrum of the ear.

However, on the ninth day following the operation, the patient began to run a daily elevation of temperature which reached 101.4° (F.). Eleven days following the mastoidectomy the amount of drainage from the wound increased and the patient developed a paralysis of the left lateral rectus muscle, a paresis of left facial nerve, and at this time we felt sure that the patient had petrositis (Gradenigo's Syndrome), but further operative procedure was delayed because we felt that the mastoid wound had been thoroughly cleaned and little could be done toward increasing the drainage, even from the petrous tip.

On the twelfth day the patient was given a transfusion of 360 cc. of citrated blood, in an effort to increase her resistance. She was again transfused on the

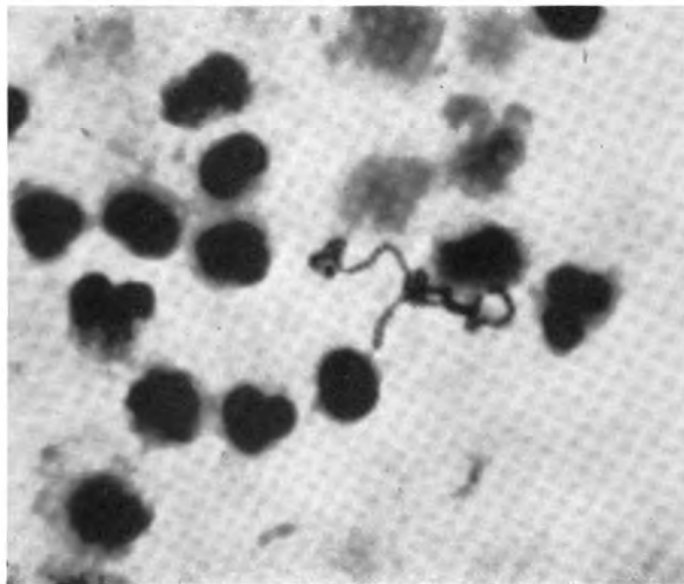


FIGURE 1.—SMEAR MADE DIRECTLY FROM SPINAL FLUID SHOWING CHAIN FORMATION LATER GROWN IN CULTURE AND IDENTIFIED AS HEMOLYTIC STREPTOCOCCUS.

fifteenth postoperative day with 400 cc. of citrated blood. On the thirteenth postoperative day a spinal puncture was done for diagnostic purposes, as the patient's condition led us to believe that we were dealing with meningitis. The spinal fluid was entirely negative, both from a morphological and cultural standpoint. At 11 o'clock on the seventeenth postoperative day the patient's temperature dropped to 96.4° (F.) to rise suddenly to 102.0° (F.) and at 5 o'clock the spinal puncture was repeated, with a withdrawal of approximately 15 cc. of turbid fluid.

Since we felt that we were dealing with a streptococcal infection, anti-streptococcal serum was ordered to be given before a complete study had been done on the spinal fluid, and we were much surprised that the fluid should show not a chain formation, but a gram negative intracellular organism which was diagnosed morphologically meningococci, to be later grown in pure culture. This finding caused us to completely reverse treatment, and the patient received anti-meningococcal serum as indicated on the table. On the following day, however, in the daily spinal puncture which was instituted, we were even more surprised to find (fig. 1) that the fluid showed an organism which both morphologically and culturally was diagnosed as hemolytic streptococci and with this picture the patient was treated as indicated by the table for both meningococcal and streptococcal meningitis.

Within 2 hours after the first positive spinal fluid had been found, the patient was carried back to the operating room and as much dura as consistent was uncovered, but the dura was not opened. Little further bone work was done except to carry the posterior wall down to almost the level of the drum and granulations were cleared from around the attic. It can be stated here that after the second operation, whether from intensive antisera treatment or from the operative procedure, the drainage from the wound had ceased entirely, and from that date on it remained clean.

On April 5 the spinal fluid cell count had dropped to 310 cells, and the patient's condition was improving in such a gratifying manner that it was decided to do an animal inoculation with spinal fluid to determine the possibility of avirulence of organisms appearing in the fluid at that time. Toward this end, a rabbit was injected intravenously with 2 cc. of the spinal fluid, and a guinea pig was injected intraperitoneally with 2 cc. of the same fluid. Both of these animals showed no reaction whatever, and remained alive and happy for three months when it was decided to use them in other laboratory work. The animals were not autopsied because we wished to give the organisms every chance to show virulence.

The patient's recovery was rapid until she developed a severe serum reaction on April 7, and for which several measures were tried, but were to no avail. Codeine and acetylsalicylic acid was given in small doses by mouth at frequent intervals to ease the extreme tenderness which the patient developed in all movable joints of the body, particularly those of the hand, wrist, elbow, shoulder, and hip. She was discharged from the hospital on April 24, 1937, and the wound was completely healed the latter part of May. Her diplopia gradually diminished, and there was a return of function to the left lateral rectus.

On this date, August 10, 1941, the patient is quite happy, has gained normal pounds of weight, and shows no residual effects whatsoever, other than a healed mastoid scar, and is leading the usual active life to be expected of young girls of her age.

SUMMARY

The following points are desired to be stressed in this case:

1. The importance of early diagnosis of Otitic Meningitis.

2. Extensive surgical exposure of dura, without opening of the same, immediately after the diagnosis of Otitic Meningitis has been made.

3. The massive dosage of antisera both intraspinally and intravenously.

4. The chemical treatment of streptococcic meningitis by use of sulfanilamide.

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RETICULUM CELL SARCOMA OF CECUM

By Commander Frederick C. Greaves, Medical Corps, United States Navy

The following case is reported because of a disease that is relatively rare and also because it presented a problem in diagnosis before surgery and a problem of pathological classification after its surgical removal.

M. A. H., a 19-year-old seaman 2c, was admitted to the hospital June 11 because of abdominal pain and discomfort of 1 month's duration. The onset of symptoms started as cramps in the lower abdomen which subsided when he eliminated solid food from his diet and subsisted on liquids. The cramps reappeared upon returning to a regular diet and became progressively more severe. In spite of the fact that he did not consider himself sick enough to go on the sick list, he felt that each day he was a little worse and during the week before admission he became nauseated and vomited each night. Anorexia and weight loss occurred. His weight on admission was 127 pounds, and this represented a loss of 35 pounds since the onset of his symptoms.

His past medical history was noncontributory and, with the exception of pneumonia and the ordinary childhood infections, he had always been vigorous and robust. His appendix was removed in June 1937, following which he made an uneventful recovery.

Weight loss was evident on physical examination. The abdomen was scaphoid and without spasticity. A well-healed scar 2½ inches long was present at McBurney's point. There was a palpable smooth, rounded mass the size of a small orange in the right lower quadrant. The mass was freely movable in all directions, was slightly tender and somewhat compressible on pressure. Peristaltic sounds could be heard throughout the abdomen. R. B. C. 5,000,000, Hb. 95 percent, W. B. C. 5,350. Segmented forms 47 percent, band forms 4 percent, lymphocytes 35 percent, monocytes 12 percent, eosinophiles 2 percent. No pathological cells were

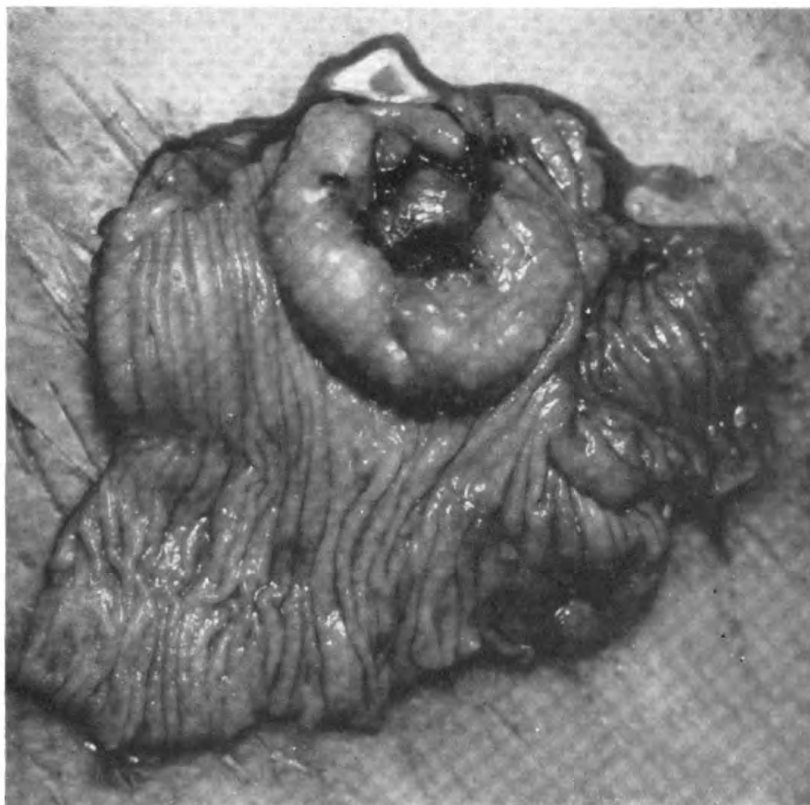


FIGURE 1.—THE SURGICAL SPECIMEN SHOWING THE TUMOR OF THE CECUM AT THE ILEO-CECAL VALVE.

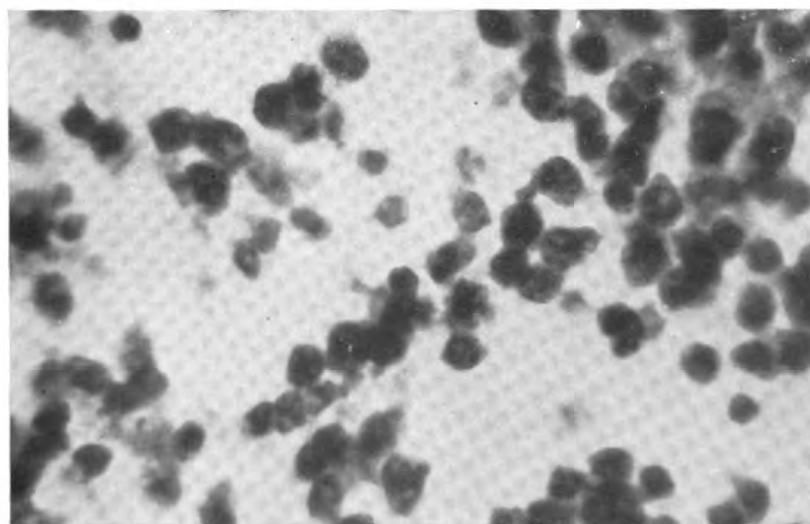


FIGURE 2.—THE MICROSCOPIC PICTURE OF THE TUMOR SHOWING HYPERCHROMATISM AND MITOTIC FIGURES.

seen. Urine normal. Blood Kahn negative. X-ray studies of the abdomen and kidney regions showed the pelvis of the right kidney to be slightly dilated and the kidney to be abnormally mobile. The right kidney region was explored surgically on June 17. It was seen that the mass was intraperitoneal, no pathology was noted in the kidney, and nephropexy was performed and the wound closed. Following this procedure it was believed that the mass receded somewhat in size. On July 1, 1938, laparotomy was performed and the mass was found to be a tumor within the cecum, close to the ileocecal valve and to be adherent to the mesentery. None of the regional lymph nodes were enlarged or appeared to be involved. The tumor was removed by resecting the ileocecal valve and adjacent portions of the ileum and cecum. The defect was repaired by a lateral anastomosis between the ileum and transverse colon. Convalescence was uneventful.

The surgical specimen, shown in figure 1, consisted of the ileocecal valve, 4 cms. of terminal ileum and 9 cms. of proximal cecum. A tumor mass measuring 5.5 cm. in diameter and 2 cms. in thickness and encroaching upon the lumen was present in the mucosa of the cecum and involved the ileocecal valve. The surface of the mass presented a depressed center with heaped up edges and was studded by numerous small white nodules resembling tubercles. There were no lymph glands present in the small amount of mesentery attached. A second lesion consisting of a shallow ulcer with irregular borders, measuring 1.0 by 0.5 cm. was present in the mucosa of the cecum on the wall opposite the tumor mass.

Microscopic examination of the tumor mass showed a tremendous proliferation of reticulum cells which made up the greatly thickened submucosa (fig. 2). Increased cellular activity was indicated by the hyperchromatism, numerous mitotic figures and infiltration of the muscle layers by these cells. The mucus membrane was intact. No multinucleated or giant cells were seen, but a few plasma cells, eosinophiles and lymphocytes were present. Acid fast bacilli could not be demonstrated. Bielschowsky's stain showed a definite reticulum increase. The smaller lesion showed an ulcer base that was subacutely inflamed and in which a rare giant cell was seen. Lymphocytes dominated the picture, only a few reticulum cells that showed no proliferative activity being present. The pathological diagnosis was reticulum cell sarcoma and tuberculous ulceration of the cecum.

DISCUSSION

The pathological diagnosis had to be made between two relatively rare conditions, namely, hypertrophic tuberculosis and lymphosarcoma. The smaller ulcerated lesion offered little difficulty. Hypertrophic tuberculosis of the intestine and lymphosarcoma of the intestine possess many histological features in common which render their differentiation difficult.

Hypertrophic tuberculosis is believed to be the reaction on the part of the tissues to tubercle bacilli in an individual who has not been sensitized previously and who possesses a strong natural immunity to the infection. Other theories have been advanced in explanation, one of which is that the lesion is the result of infection by the bovine type of organism. Another, that it represents the exposure to a small number of bacilli or to bacilli of attenuated virulence.

The histological picture closely resembles that seen in an initial tuberculous infection. It is a slow productive process with very little

tendency to ulcer formation. This feature is in marked contrast to the picture present in the more common form of multiple tuberculous ulceration of the intestine. Tumefaction occurs as the result of cellular proliferation in the submucosa and the lumen is encroached upon. Giant cells are usually present in large numbers, but they may be absent entirely. Tubercle bacilli are not so readily demonstrated. The regional lymph nodes enlarge and constitute a prominent feature of the disease. The site of predilection is at or near the ileocecal valve, although the lesions may be encountered in any part of the intestinal tract. The disease is most common in early adult life.

The preoperative diagnosis is made with difficulty. The symptoms complained of are indefinite abdominal distress with varying degrees of intestinal obstruction, depending upon the size and location of the mass. The tumor often is palpable and its presence is further confirmed by x-ray studies.

Primary lymphosarcoma of the intestine is one of the rarer forms of neoplasm. Mayo and Robbins (1) reviewed the literature in 1935 and reached this conclusion. Both Boyd (2) and Ewing (3) call attention to the difficulties attending the diagnosis of the condition.

Tumors of the lymphoid tissue are always difficult to diagnose. This is due largely to the fact that it is a composite tissue made up of different elements, any one of which may undergo neoplastic change. It is also due to its function of a filter which causes it to mirror all the irritative factors that occur upstream in its drainage area. It is composed of pure lymphoid elements commonly recognized as lymphocytes and of the reticulo-endothelial tissues which form the skeletal framework upon which the lymphoid tissue depends for support. The reticulo-endothelial tissue can be divided into the reticulum cells and the endothelial cells lining the lymph spaces. The reticulum cells are somewhat larger than the ordinary lymphocyte with large vesicular, eccentrically placed nuclei. They take part in the generalized hyperplasia that accompanies an adenitis and they are actively engaged in the changes that occur in tuberculosis. However, in both these conditions their hyperplasia is moderate and conservative, they remain orderly in size and shape, hyperchromatism is infrequent and active mitosis rare.

When primary neoplasm occurs in lymphoid tissue it usually affects only one of the component parts. It will be a lymphosarcoma but the tissue involved may be the lymphocytes or it may be the reticulum or the endothelial cells. In any event, the cells concerned proliferate and wipe out the other elements, they invade the capsule and metastasize. In the case reported above the reticulum cells are the offenders. Reticulum cell lymphosarcoma, in addition to the usual signs of anaplastic cellular proliferation, also shows an increase both in size and density of the reticulum fibers which are products

of the cells themselves. The cells often resemble berries growing on a stem of a vine. The sections show none of the giant cells seen in a tuberculous process nor the multinucleated cells of Dorothy Reed which are so characteristic of Hodgkin's Disease.

The prognosis in lymphosarcoma is poor for permanent cure. It is a common observation that the lymphocytic cell lymphosarcoma is very readily affected by the x-ray. These tumors in the cervical axillary and mediastinal area can be made to diminish in size so that they no longer may be demonstrated, but the fact remains they practically all recur either locally, or at a distance, or both. In the present case the original tumor was completely removed, the regional glands did not appear affected and the patient has been the recipient of intensive x-ray therapy. Two months after operation he has no symptoms and shows no evidence of local or metastatic recurrence. It is hoped that the neoplastic process was a very early one and that its complete removal and post-operative x-ray therapy will effect a cure.

SUMMARY

1. A case of early reticulum cell sarcoma of the cecum is reported.
2. A short pathological discussion of lymphosarcoma and hypertrophic tuberculosis is also given.

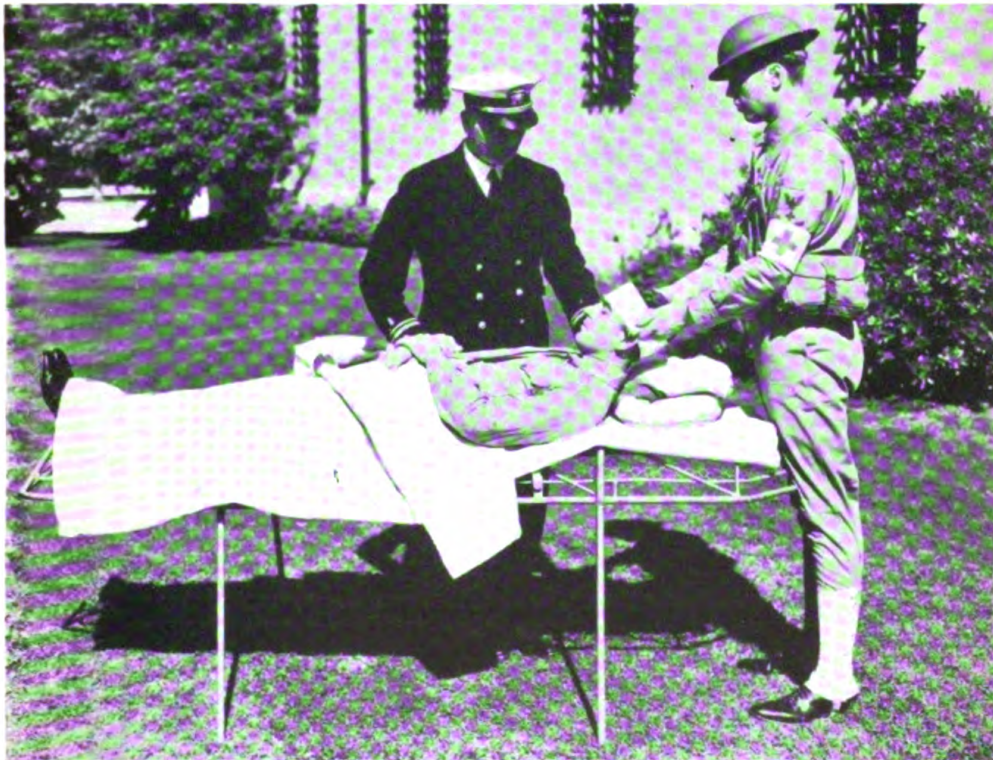
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EXPERIMENTAL LITTER IN SECTIONS.

Demonstrating lightness in weight. The foot-half of litter shows experimental folding type of field transportation arm and leg splints—as temporary fittings of litter, held in position between webbing wires and frame struts. These splints accompany litter so that they may be applied to fracture of extremity before any attempt is made to place patient in litter.



By use of simple attachments, with four stanchions, a splint litter, inverted, can be improvised as a dressing table.

562-1



RIGHT AND LEFT EXPERIMENTAL POUCHES UNROLLED AND SNAPPED TOGETHER TO FORM A FIRST-AID-APRON, WITH ALL ARTICLES ACCESSIBLE AND NEATLY STOWED IN POCKETS.

Note the straps used to secure the right and left experimental pouches, when rolled, are buckled together to form a sling which suspends apron-pouch from hospital corpsman's neck, and the other straps secure apron-pouch to the waist.

MEDICAL AND SURGICAL DEVICES

EXPERIMENTAL FIELD MEDICAL EQUIPMENT

By Captain William L. Mann, Medical Corps, United States Navy, and Chief Pharmacist
Archie B. Brown, United States Navy

The writers of this article have initiated some experiments in designing newer field medical devices, which are illustrated in this article. These experiments were conducted with a view to further "streamlining" the expeditionary equipment designed for the field forces of the naval service.

EXPERIMENTAL LITTER, METAL

This model litter is constructed of aluminum alloy which decreases the weight to nine pounds in comparison to about 25-pound weight of the Stokes stretcher.

The absence of the perineal ridge further decreases the weight and makes possible sufficient space within the litter for the application of a "Thomas" type leg splint, which is difficult in the type of litter currently available.

Previous experience demonstrates that the mortality rate from fractures of the lower extremities is decreased if a traction splint is applied before moving patient to a litter.

However, in the event that a traction leg splint is not available, counter-traction is obtained by the use of straps crossing each other at the perineum and attached to the litter frame. For practical purposes these straps secure the patient in a similar manner as does the perineal ridge when the loaded litter is hoisted in an upright position.

Stowage and individual handling is facilitated by virtue of the sectional feature permitting it to be stowed in a compartment half the litter's length. The disjointable feature provides a piece of equipment that can be carried on landing operations or on the maneuver in a manner similar to any other "back-pack," thus leaving the hospital corpsman's hands free for use in embarking, debarking, climbing over rugged terrain, et cetera.

FIRST-AID APRON POUCH

The main features of the first-aid apron pouch are: (a) accessibility of contents when the pouch is unrolled, and (b) the dual use as a pack container and as an apron with pockets.

The pockets of shallow depth give visual location of any article within and do not require the aid-man to forage to the bottom of a deep pouch for such article or the dumping of entire contents to locate the needed article at the bottom of the pouch. When use is required at a permanent location the left and right pouches can be fastened together and worn as an apron, with all items readily accessible.

FOLDING TRANSPORTATION SPLINTS

These splints are modified from and give similar traction as the Thomas splint. They combine simplicity of design, minimum weight, adequate strength, and compact storage.

Their folding feature and light weight make them particularly adaptable to field use. The design of rubber padding for the rings gives more comfort to the patient and the web strapping and slots make for more permanency of traction without risk of slipping.

The final improvements in splint devices were made in collaboration with Colonel Ralph Kaysen, Medical Reserve, United States Army, and have been more fully described in a previous issue of the Military Surgeon.

It is realized that the experimental devices are still in the developmental stage, and before adoption as standard articles of equipment, improvement with changes in details of construction will be required after a series of field tests.

The purpose of this article is to present the basic ideas.

ANKLE STRAPPING

By Lieutenant Commander G. D. Delprat, Medical Corps, United States Naval Reserve

Sprains of the ankle joint are among the most common injuries seen in every-day practice. Almost invariably, the ligament injured is the external lateral ligament of the ankle, extending from the lower end of the fibula to the astragalus and calcaneus. Sprains of this ligament result in swelling and discoloration, and if improperly treated result in a weak ankle. The injury is usually sustained on forced eversion of the ankle.

The method of strapping an ankle, as described below, was adopted by Dr. Alanson Weeks 30 years ago, and has been used as a routine in this office for the treatment of these injuries since that time. Because so few textbooks on orthopedics illustrate satisfactory strapping of an ankle joint, and because we have seen so many ankles that have been improperly supported for an injury of this kind, it would seem worthwhile to present these illustrations and bring them before the medical profession.

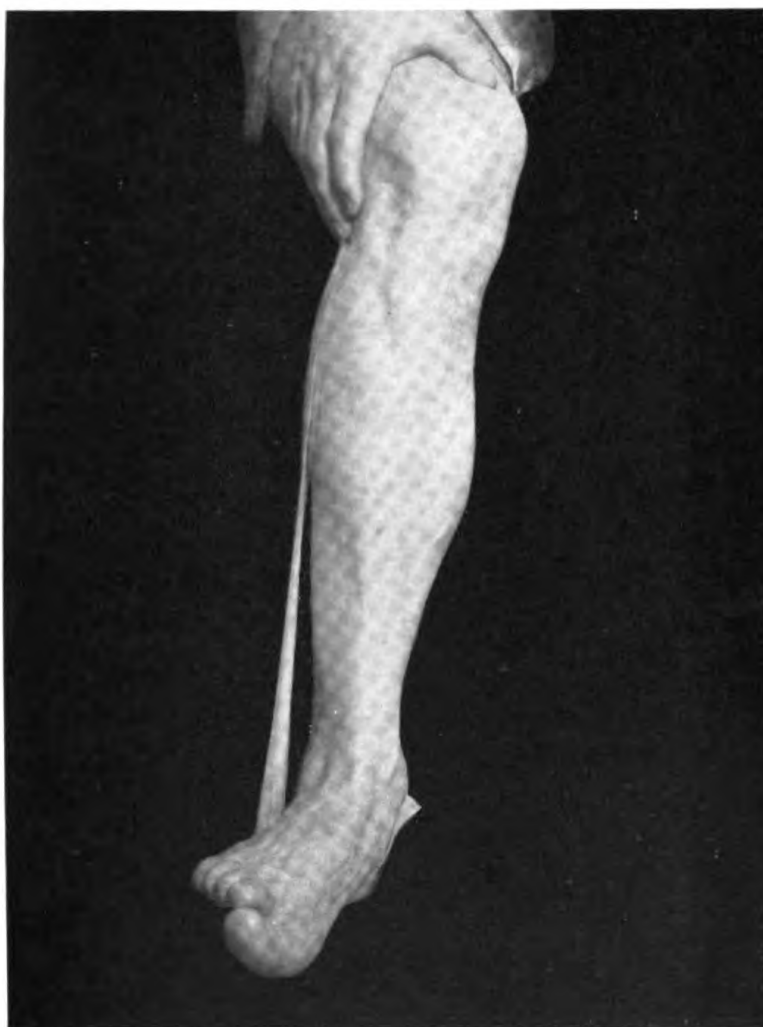


FIGURE 1.—TWO-INCH ADHESIVE STRIP APPLIED COMMENCING JUST BELOW THE INTERNAL MALLEOLUS, CARRIED AROUND THE SOLE TO THE HEAD OF THE FIBULA AND HELD WITH TENSION, HOLDING THE ANKLE IN EVERSION.

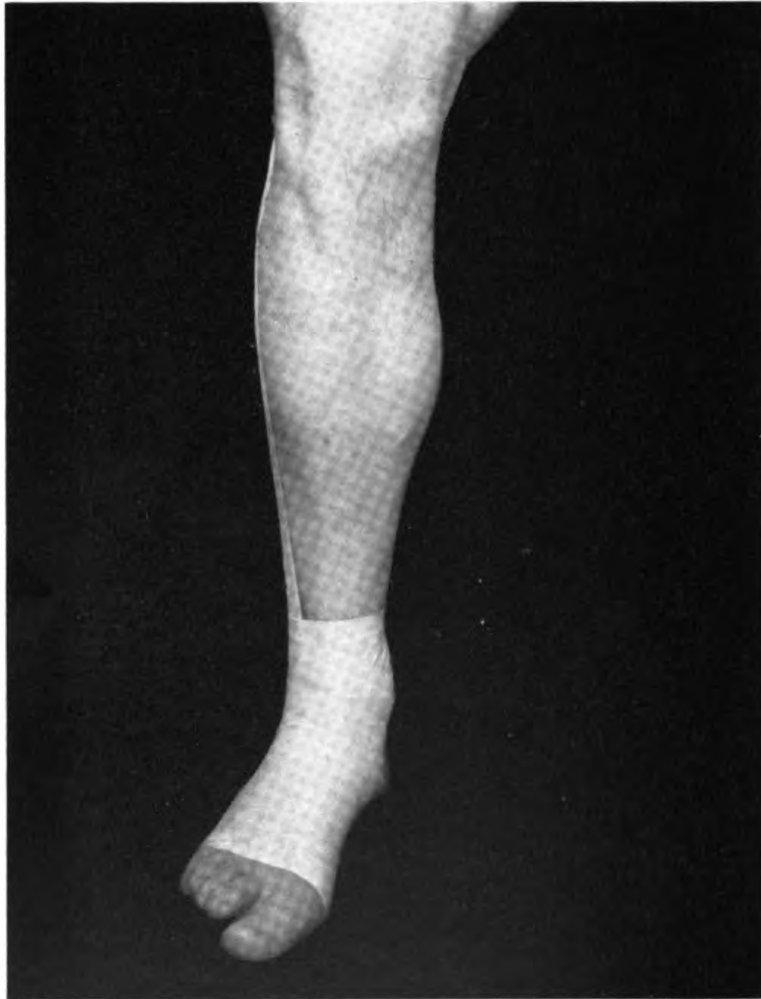


FIGURE 2.—TO PREVENT EDEMA BELOW CIRCULAR STRIP FURTHER ADHESIVE BANDS ARE APPLIED ABOUT THE ANKLE ALSO LEAVING A FREE SPACE ALONG ACHILLES TENDON AND ALONG THE SOLE OF THE FOOT.

The adhesive strapping is applied in the following manner:

After carefully shaving the leg from the knee to the ankle, a 2-inch strip of adhesive of sufficient length is applied, commencing just below the internal malleolus, is passed across the sole of the heel and held in close apposition to the head of the fibula. At the same time the ankle is held at flexion to a right angle, and with as much eversion of the foot as is possible to obtain. (See fig. 1.)

The adhesive is then applied to the side of the leg, under tension, and held by the patient's fingers at the head of the fibula.

A second strip of adhesive is then applied, holding the adhesive in apposition to the leg at the region of the external malleolus. This strip does not circle the leg, but leaves a space one inch or more wide over the Achilles tendon.

Theoretically, this support should be sufficient to splint the torn external lateral ligament, but actually if the procedure is stopped at this point, edema will occur over the foot distal to the circular strip. To prevent the formation of this edema, further strips of adhesive are applied surrounding the foot from the fold at the toes up to the first circular strip applied. (See fig. 2.) All these strips, however, leave a space along the sole of the foot, and none of them overlap or press upon the Achilles tendon.

A circular bandage is finally applied, serving to hold the adhesive in tight apposition to the skin, which is left in place for 24 hours. The adhesive is left on as long as 10 days if the skin tolerates the presence of the plaster, at the end of which time it may be replaced if necessary. On some individuals the entire dressing is changed after 4 or 5 days if it seems that the skin does not well tolerate the adhesive.

The advantages of this type of strapping are obvious. It serves to support the torn external lateral ligament by means of a wide strong strip of adhesive extending as far up the lower leg as possible, and therefore capable of taking up the strain and preventing the ankle being turned outward. The freedom of the Achilles tendon is very important, and no circular bandage should ever be applied which presses upon it.

Even with severely injured ankles and marked swelling and pain it has been possible to return men to active work immediately after the application of the above strapping. Such results are certainly not possible with the narrow overlapping straps frequently applied about the ankle joint.

EYE PROTECTION FOR AVIATORS

By Lieutenant Stephen E. Flynn, Medical Corps, United States Navy

One of the most important physical examinations for entrance into the Naval Academy and the Navy is the rigid examination of the eye.

The visual acuity of all applicants must not fall below 20/20. More applicants fail in the eye examination than in any other portion of the physical examination. Many young men have been greatly disappointed after much preparation for his entrance examination into Annapolis only to find that he has failed because of defective vision.

Very little has been done in the service toward safeguarding normal vision, which is probably one of the most important factors in the making of a well-qualified officer or man and which is especially true of those in aviation.

Many forms of tinted lens are being used by pilots and other personnel connected with flying to protect the eyes from the glare emanating from clouds, or when flying directly into the sun. In examining several hundred flyers and personnel connected with flying, all have admitted that tinted lens cause their eyes to burn and smart, plus a drawing sensation about the eyes when the glasses are worn for any length of time, and that the longer the tinted glasses are worn, the less bright sunlight can be tolerated.

According to Ebert (1) and Boyden (2) the eyes have a perfect physiological shutter system which regulates the amount of light rays allowed to fall on the retina. This shutter system is actuated by two sets of muscles which receive stimuli from the amount of light admitted. In placing tinted lenses in front of the eye and shutting out one or more portions of the visible spectrum, these muscles become atrophied, which, because of non-use, causes the visual system to become increasingly sensitive to, and increasingly unable to cope with light. In short, tinted lens acts as a crutch to the shutter system.

Many people wear tinted lens for cosmetic reasons to eliminate frowning, squinting, etc., which in turn cause excessive lines and wrinkles in the face, but few seem to realize that the eyes become weaker and that lines are more apt to form after the glasses are removed. The only one who receives much benefit from tinted lenses is the optician who sells them and the advertising agency that puts them on the market.

No other organs of the human body that play such a responsible role in life receive such little consideration as do the eyes. The greater portion of the public, who have poor vision, will consult the advertising doctor who sells glasses, but who is not qualified to fit glasses, and many will buy their reading glasses from the trial case in the 10-cent store.

Many times aviators are compelled to fly into the sun above the clouds where the glare from the clouds and the sun is intense. This excessive amount of illumination will produce a deleterious effect upon the eye structures, especially upon the lens and later the retina. According to Metcalfe (3) the supply of pigment of the retina is dependent upon light which stimulates the visual purple, but excessive



SIDE VIEW SHOWING SHADED EYES WITH EARPHONES EASILY APPLIED.

amounts of light, such as sun glare for long periods on the retina, will cause destruction of the pigment, which is not regenerated until the excessive illumination is removed or diminished

While tinted lenses have been found to be wholly inadequate for eye protection among flight personnel, a cap similar to a baseball cap, having a long visor, has been utilized by a few of the flight personnel on duty at the United States Naval Air Station, San Diego, Calif., with satisfactory results. The cap is form fitting, blue in color, and has a long visor, the underside of which is green in color (fig. 1). Because of its form-fitting qualities it is not easily blown off and doesn't interfere with the wearing of head phones. This type cap may also substitute for the regulation white hat now worn by members of the plane-handling crews aboard aircraft carriers, which, in addition to being difficult to retain on the head in a wind also creates a glare hazard to pilots approaching the flight deck for landing. Because it is form fitting it does not squeeze the scalp around the hat band cutting off or diminishing the blood supply to the top portion of the scalp and interfering with the nourishment to the hair follicles which increases the tendency toward baldness.

SUMMARY AND CONCLUSION

1. That many types of tinted glasses are being used with harmful effects.
2. That excessive illumination to the eye prolongs recovery time, due to the destruction of visual purple which adds to the hazard of night flying.
3. That the new type of visor is suggested to eliminate excess light which is harmful and injurious to the retina.

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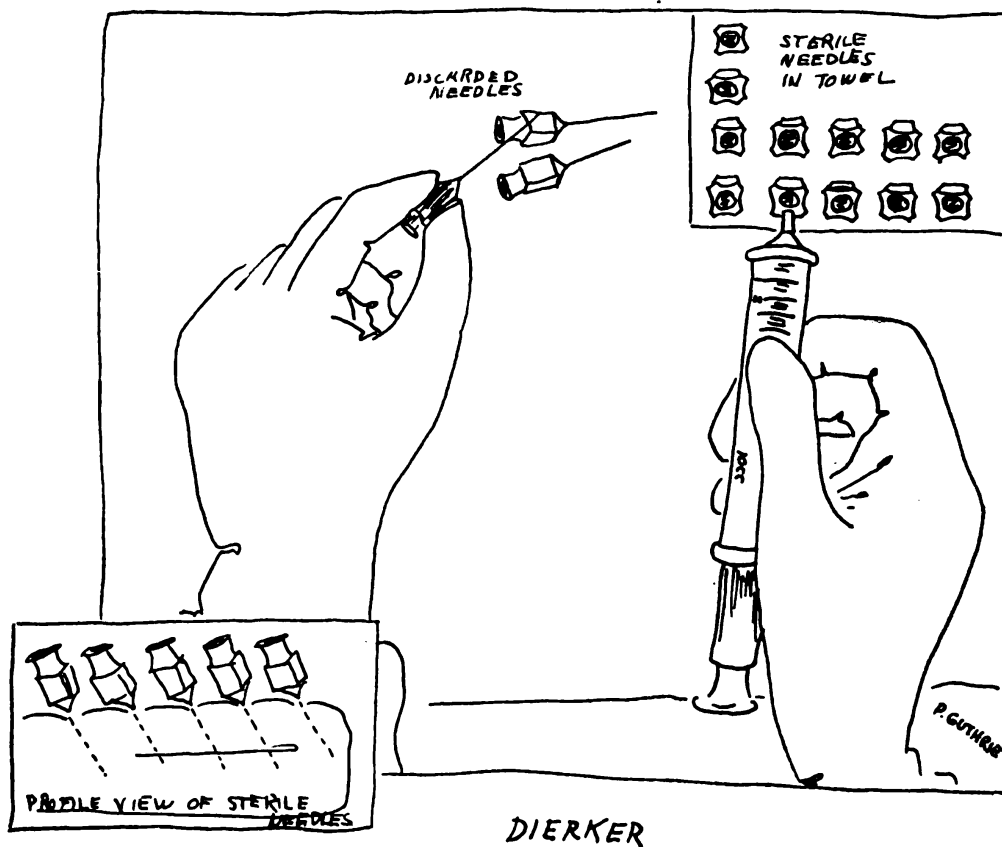
1. Ebert, E. C.: Eyestrain. U. S. Nav. Med. Bull. July 1940.
2. Boyden, R. C.: Ophthalmological facts, fads, and fallacies. U. S. Nav. Med. Bull, July 1940.
3. Metcalfe, E. E.: New method for determining night blindness. U. S. Nav. Med. Bull., April 1940.

A METHOD OF PREPARING HYPODERMIC NEEDLES FOR MULTIPLE INJECTIONS

This method of preparing hypodermic needles was devised in order to facilitate speed while giving multiple hypodermic injections. The accompanying diagram illustrates this method which was submitted by Hugh Dierker, M. D., Assistant District Health Officer, of the County of Los Angeles, Calif.

A "bath towel" is folded around a piece of heavy cardboard in such a manner as to leave several thicknesses of the towel over the

cardboard and to present a smooth surface. Into this the "hypo" needles are inserted in rows at a 45° angle. The whole is enfolded in a hand towel, or muslin, and sterilized in the autoclave. When taken into the field, this is placed at the operator's side, and in using a large amount of material in a single syringe it is unnecessary when changing needles to use forceps, search for needles, replace forceps, and tighten needles. All that is necessary is to insert the hub of the syringe into the needle and enough friction exists between needle and hub to allow withdrawal of the needle from the loose mesh of the



DIERKER

FIGURE 1.—Dierker.

toweling. The needle can be tightened between thumb and forefinger without contaminating the point or shaft, and it can be removed by hand and discarded while the syringe is picking up another needle.

Advantages.—1. Eliminates many motions in changing needles.

2. Low cost. (a) It requires no more time to prepare the needles in this manner because the needles must be cleaned after use anyway and to insert them in a towel is very nearly as simple as dropping them. (b) No special apparatus is required.

3. Life of needles prolonged due to protection of points.

4. Increases speed and efficiency in the field with diminution of fatigue.

METHOD FOR EMERGENCY BLOOD TRANSFUSION DEvised IN RUSSIA

A novel and unique method of giving blood transfusions just behind combat lines was that devised by Dr. W. Dsbanowsky, who describes this method in a Russian medical journal published in the Ukraine, the *Radianska Meditzina*, No. 7-8, 1940, page 78-79.

The method described is very simple and, after having transfused several hundred wounded on the battlefield, its application has been highly satisfactory.

A child's ordinary rubber ball is used, having the capacity for at least 500 cc. of fluid. A wire loop is made to fit around the ball in order to control this distension and thus its capacity. The diameter of this loop was previously measured. One or more loops may be used depending upon the amount of blood to be transfused. A flexible rubber hose, two-way cock, and needles are all that is necessary. This material is previously sterilized and stored by wrapping in a sterile towel.

When giving a transfusion, the operator sucks through this apparatus 5 percent sodium citrate several times until all surfaces are well lubricated with the solution, keeping about 50 cc. in the bulb. The bulb is compressed allowing the air to be expelled through one opening of the petcock. The handle of the cock is then turned, preventing the return of air into the ball and permitting the expulsion of air from the tube. The needle is then inserted into the vein, the ball is lowered, and air is permitted to escape from the hose through the cock until the blood is seen, when the cock is turned permitting the blood to enter the bulb both from gravity and from suction produced by the expanding walls of the ball. When the ball has expanded to the capacity permitted by the wire loop, the cock is again turned, the needle is withdrawn, changed, and placed into the recipient. The ball is then elevated and the return of flow can be controlled both from gravity and by compression on the bulb. When all blood has entered the recipient the cock is turned, needle withdrawn, and apparatus cleaned and made ready for another transfusion.

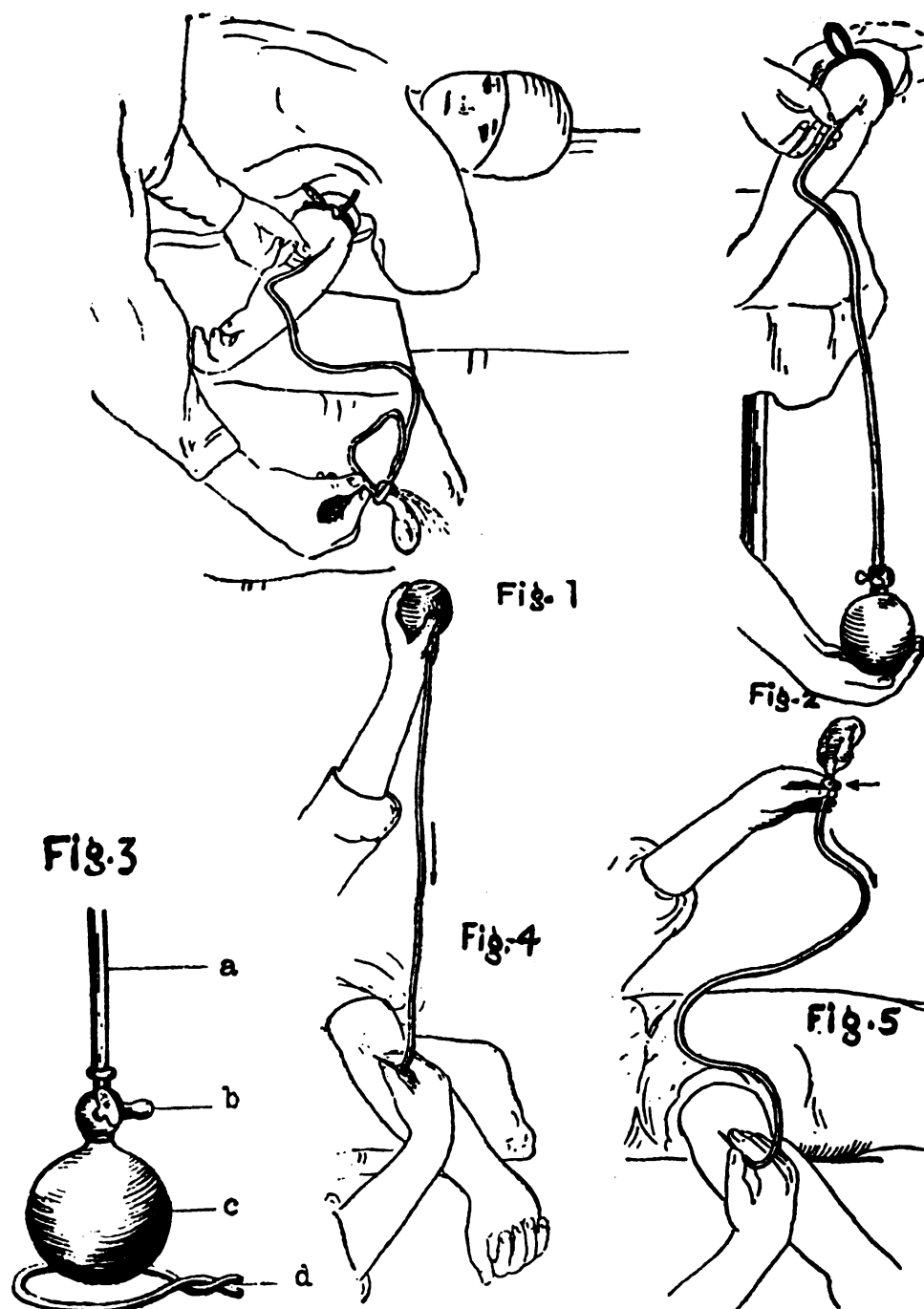


FIGURE 1.—Insertion of needle into donor. Air escaping from tube.

FIGURE 2.—Expansion of the walls of rubber ball and gravity forces blood into ball.

FIGURE 3.—(a) Rubber tube; (b) two-way stop cock; (c) rubber ball; (d) wire loop for regulating the volume of blood.

FIGURE 4.—Transfusing the blood into the vein of the recipient.

FIGURE 5.—End of transfusion showing collapsed ball.

NOTES AND COMMENTS

PHYSICAL FITNESS OF OFFICERS

Because of the rigid physical standards for entrance and the annual physical examinations given to all officers of the Navy, they form a highly selected group from a health standpoint. Despite this fact, unceasing attention must be given to the maintenance of the health of officer personnel not only by the Medical Department, but by the officers themselves.

One of the subjects to which special attention should be directed is that of exercise and recreation. This must be individual in character to be of real use and, indeed, to prevent it being detrimental. The ages of officers on the active list of the Navy cover a period from 20 years to 64 years of age. It is obvious that the exercises suitable to the lower age group are not only unsuitable but apt to be harmful to the higher age group. It behooves every officer to give serious thought to the question of his own physical well-being on which his efficiency so much depends; to regard it as a matter of real importance and to give to it at least as much time as he would give to keeping his automobile in good running condition. The exercises to be selected should be those available, practicable, and suited to the officer's needs. The exercise may vary from active participation in athletics among younger officers, to golf or gardening among the older ones. The availability of exercise must also be thought of by the naval officer who may be at sea and not have the facilities which may be at hand when ashore. A few minutes of setting-up exercises, walks on the deck, volley ball, deck tennis, shuffleboard, or similar games may then be used to maintain the tone of muscles.

A strong influence on health is the effect of recreation, by which is not necessarily meant physical exercise but any diversion or employment which breaks the monotony of the regular tasks and routine. Seeing a moving picture, listening to music, reading a book, or even resting are in a sense recreations and their effect on health must not be forgotten.

POSTGRADUATE INSTRUCTION FOR MEDICAL OFFICERS

The attention of medical officers, both Regular and Reserve, is invited to the fact that several vacancies exist in the following Medical Department specialties: Aviation medicine (Regular Navy), leading to the designation of flight surgeon; aviation medicine (Reserve officers only)

leading to designation, aviation medical examiner; x-ray; psychiatry; internal medicine; surgery; deep-sea diving; and eye, ear, nose, and throat.

The Bureau will be pleased to receive official applications for postgraduate instruction in the above-mentioned specialties from medical officers of the Regular Service and of the Naval Reserve on active duty. Applications should be forwarded immediately to the Bureau of Medicine and Surgery, via official channels, and in accordance with Bureau circular letter M, appendix D, Manual of the Medical Department, United States Navy.

Commanding officers or the senior medical officers of the stations to which the applicants are attached are requested to comment fully on all requests for postgraduate instruction.

As a general rule, medical officers who have been designated for postgraduate instruction in specialties other than those pertaining to aviation and deep diving are assigned to instruction at the major naval hospitals, and if they demonstrate particular adaptability in their specialty are encouraged to submit a further request for continued instruction at civilian medical centers. Advanced training in flight surgery and aviation medicine is given at the Naval Air Station, Pensacola, Fla.; that in deep diving at the Navy Yard, Washington, D. C.

IMMUNIZATION WITH TETANUS TOXOID AND YELLOW FEVER VACCINE

It is 30 years since the Navy, by the introduction of the use of antityphoid inoculation, practically eliminated typhoid fever among naval personnel.

Smallpox vaccination was used in our Navy probably as early as 1800. The first authentic record was in 1808 and in 1848 it was directed that all recruits at recruiting depots be vaccinated before being sent to ships or stations. It is evident from this that the introduction of the general use of a new vaccine for all naval personnel is not an every-day occurrence, but is something of an important event.

The inoculation against tetanus with alum precipitated tetanus toxoid and vaccination against yellow fever are measures worthy of note. Directions have been issued for all personnel to be immunized against tetanus and yellow fever. Definite instructions as to the administration of the vaccines and the record to be made of them have been issued by the Bureau of Medicine and Surgery. Tetanus is one of the most important diseases in the military service in times of either peace or war. The fact that the Navy operates in all parts of the world and may often be in areas where yellow fever is endemic makes vaccination against this disease of great value to naval personnel.

VALUE OF MINIATURE ROENTGENOGRAPHY

The use of miniature roentgenography for the detection of tuberculosis in the examination of recruits for the United States Navy is beginning to prove its value. This is believed to be the major factor in the decrease of Navy personnel hospitalized for tuberculosis, and the number of early cases of tuberculosis found in the new applicants, since this method of examination was adopted, and which the stethoscope failed to detect. Not only have the miniature photographs picked up these undetected cases of tuberculosis, but they have also brought out cases of heart disease, new growths of the lungs, aneurysms of the aorta, emphysema and silicosis.

This method has not only proved valuable in that it is an economical procedure for mass examinations, but in detecting early tuberculosis it is doing away with the "carrier" who contaminates others before he is detected.

The United States Navy was one of the pioneers in the development of the use of miniature roentgenography as a practical and economical procedure for the detection of early tuberculosis. This method was developed as a result of the demand for a procedure by which large numbers of men could be examined rapidly and with far less cost than by the usual roentgenographic examinations. Since this method has been perfected, its value has been recognized by other countries and it is now being used by England and Germany, and probably by Italy.

THE IMPORTANCE OF MALARIA

The importance of a disease may be measured by its prevalence, its duration, its death rate, and the disabling effects of its sequelae. The common cold has a wide prevalence and it is estimated that every person in the country has probably two colds a year. Fortunately, the course is usually short and while serious results may occur from the sequelae, the death rate and disability rate is really low. On the other hand, such a disease as diabetes occurs in relatively few individuals. Its death rate is low but the damage due to partial invalidism is considerable. In certain other diseases, such as epidemic meningitis, the incidence is not great but the death rate is very high. A few diseases combine a high incidence, high death rate, and a high rate of invalidism. The most important of these is malaria.

In the report by the health organization of the League of Nations, quoted by Stitt in the new and as yet unprinted edition of his *Diagnostics and Treatment of Tropical Diseases*, it was estimated that 17,750,760 cases of malaria were treated in 65 countries during one year. Yet this represents only a small number of the cases of malaria existing in the world, for it was estimated by the Commis-

sion that even in India where organized effort is constantly being made to extend medical treatment to all cases, there were 100,000,000 malarial cases. Probably not more than 8,000,000 or 10,000,000 received treatment. India is the largest of the malarial countries but it is estimated that the whole of Africa, except certain highland areas, is affected and that the number of cases runs into millions annually. In the Western Hemisphere in great parts of Central and South America, malaria is a cause of great mortality.

Here is a disease, then, which perhaps affects more people than any other disease, with the exception of the common cold, which often has a high death rate and also produces prolonged periods of disability. It is not surprising, in view of these facts, that malaria has been considered as a primary factor in the fall of some of the ancient empires, or that students of economics have ascribed the material poverty of many countries to this disease.

The naval medical officer, because of his service in tropical possessions, is particularly interested in malaria and everything in regard to its history, its diagnosis, its symptoms, and its treatment, should engage his serious attention and study. This is more than ever the case now when new Caribbean bases, all in the malarial zone, are being occupied.

CONCENTRATION OF SULFANILAMIDE IN THE AQUEOUS HUMOR OF HUMAN EYES

T. H. Luo and S. Y. P'an, from the Departments of Ophthalmology and Pharmacology, Peiping Union Medical College, Peiping, China, in their paper published in the Chinese Medical Journal of August 1940, stated that they used the aqueous humor from the eyes of patients to determine the concentration of sulfanilamide. The experiments were carried out on patients with greatly impaired vision. The drug was used orally, by local instillation of a 1-percent solution, and by local application.

When the drug was given orally and in divided doses, the first puncture of the anterior chamber was performed 2 to 3 hours after the last dose, and the second, 1 hour after the first. Immediately following each aspiration, blood was obtained for sulfanilamide determinations. For local instillations, 1 percent of a warm solution of sulfanilamide was employed. When used as a local application, one instillation of a 1-percent solution of "pantocaine" preceded the finely divided sulfanilamide, which was applied over the conjunctival and corneal surfaces until a thick layer had formed. The eye was kept closed for 1 hour before aspiration. Before each paracentesis the eye was thoroughly irrigated with a sterile solution of normal saline.

The results obtained showed that sulfanilamide was found in the aqueous humor after all three methods of administration, not only

in the first specimen, but also in the second and third specimens obtained from 1 to 3 hours after its administration. However, the most significant finding was the high concentration of the drug following local application in the form of powder, in comparison to the large doses taken orally. There were no reactions in the eyes after local application of the drug. The results indicated that the local application of sulfanilamide deserved further trial in the therapy of ocular infections.

ACCIDENT PREVENTION REMINDERS FOR HOSPITALS

The following are some accident prevention notes from an article in *Modern Hospital*.¹ Have you—

- Taught your personnel to report all worn or broken equipment?
- Reminded your employees to turn on lights before entering dark rooms?
- Prevented falls by insisting that all dropped leafy vegetables be picked up immediately?
- Required that only one-half of the corridor be washed or waxed at a time?
- Provided a portable sign on a stand, 'Danger—floor being waxed'?
- Furnished your laboratory workers with leather gloves to be used when handling laboratory animals?
- Posted signs outside the operating room, warning of explosive gases in use?
- Admonished your nurses not to discard surgical blades, razor blades, and needles with the soiled linen?
- Advised your nurses and laboratory workers against storing bottles of alkalis and acids on shelves above eye level, or beyond reach?
- Educated nurses in proper use of a towel and water when inserting glass tubing into rubber stoppers or tubing?
- Taught porters to empty waste baskets by inverting them, instead of reaching into them with their hands?
- Replaced worn stairs, or made them slip-proof with strips of carborundum?
- Noticed how often accidents occur as a result of inattention to work?

CONDUCTIVITY OF TERRAZZO FLOORS

Static electric charges which occur in operating suites must be carried off to a suitable ground, if anesthetic explosions are to be avoided. Experiments were conducted to determine whether metal grilles embedded in terrazzo floors were dependable for this purpose. It was found that "brass mesh embedded in the terrazzo will not form a ground of sufficiently low resistance to be relied upon for the satisfactory carrying off of static charges * * *. The only satisfactory ground was obtained where contact was made directly with the exposed metal."

It is recommended that brass grilles 4 by 4 inches be used, instead

¹ Katzlve, J. A., *Modern Hospital*, May 1941, p. 76.

of those with larger spaces. The metal should not be less than one-sixteenth inch thick, with all joints soldered. It should be grounded through a suitable, known resistance, which is accessible for frequent testing and replacement.

THE SLEEPING POSTURE AND NEUROCIRCULATORY ASTHENIA

Effort syndrome, irritable heart, soldier's heart, and orthostatic tachycardia are all names for familiar clinical conditions for which perhaps the best term is neurocirculatory asthenia. It is a condition of great importance in the military service because of its high incidence, particularly among recruits. Any new light thrown upon it commands the attention of all military medical men. Recent research carried on at the Mayo Clinic, Rochester, Minnesota, is therefore of importance.

It was found that in orthostatic hypotension there was great improvement if the patient slept with the head of the bed elevated. This was found particularly useful with patients with secondary orthostatic hypotension following an operative procedure or debilitating illness.

It is a striking fact that many cases showing symptoms of effort syndrome, frequently point out that they are worse in the morning and it takes them 2 or 3 hours to "get going." It would appear that part of this, at least, is due to the length of time required for the vasomotor system to adapt itself to an abrupt change from the horizontal to the vertical position. In other words, it is a question of physiological adjustment to gravitational hydrostatics. It was found by plethysmographic measurements that more than a pint of blood was retained in the lower extremities when the erect posture was assumed after a period in the horizontal position.

Where the patient was placed in a slanting position during the night, the change in posture on assuming the erect position in the morning was not so great and the adaptation to be made was less severe. At Rochester, Minnesota, MacLean, Magath, and Allen found that a number of their cases of primary orthostatic hypotension who were unable to remain in the erect posture for even a moment were able to be up and carry on some of their ordinary activities.

As we know from many anatomical facts, man has not yet fully become adapted to the upright position. He now lives in both the vertical and horizontal positions; the former during activity, and the latter during sleep. It is not surprising, therefore, that his circulatory system is not adapted to abrupt changes between these two planes.

These studies indicate a method of treatment for neurocirculatory asthenia which is simple, harmless, and easy of application, and which promises to be of great value in these troublesome cases.

**REQUEST FOR THE RETURN OF OLD COPIES OF THE U. S. NAVAL MEDICAL
BULLETIN**

There has always been a great demand for copies of both the current and early numbers of the **BULLETIN**, in this country and in many foreign countries. For a long time the **UNITED STATES NAVAL MEDICAL BULLETIN** was the only journal of naval medicine published in the Western Hemisphere. Many medical libraries and research centers are particularly anxious to obtain it. The number of copies printed exceeds by only a few the number required by the regular circulation list and, consequently, there are limited copies available to supply the requests which are being frequently received.

If, therefore, anyone receiving the **BULLETIN** regularly would return any copies they do not wish to keep, the Editor would be glad to receive them. They may be forwarded by placing them in a franked envelope. On the back of the title page of the **BULLETIN** are listed some numbers which are particularly required, but any copy from the first in April 1907 to the last issue can be used.

BOOK NOTICES

Publishers submitting books for review are requested to address them as follows:

The Editor,

UNITED STATES NAVAL MEDICAL BULLETIN,
Bureau of Medicine and Surgery, Navy Department,
Washington, D. C.

(For review)

FIELD SURGERY IN TOTAL WAR by *Douglas W. Jolly, M. B., Ch. B. N. Z., late Major, Spanish Republican Army Medical Service.* With a Foreword by *Surgeon Rear Admiral G. Gordon-Taylor, O. B. E., M. A., F. R. C. S., F. R. A. C. S.* Pp. 242. Paul B. Hoeber, Inc., New York, publishers, 1941. Price \$3.50

The word "timely" is apt to be overworked in these days but it can certainly be applied to this little book in which an eminent English surgeon has embodied his experiences in the Spanish Civil War. From personal experiences he describes the tactical employment of a mobile hospital in the field and the treatment of some 5,000 wounded. Of particular interest to the military medical man is his description of the handling of the wounded in the field by what was called the "three-point forward system." This consisted of a casualty classification post for the brigade or division, a number one hospital for the most urgent cases, and a number two hospital for less urgent cases. The writer emphasizes the importance of early surgical intervention in all abdominal wounds and wounds with severe hemorrhage and shock. He describes well the use of underground hospitals established in great natural caves or in railway tunnels. The difficulty of caring for the wounded under war and battle conditions, especially in the face of air raids, is clearly shown. The effect of climate and weather not only in all military operations but in the evacuation and care of wounded is emphasized, particularly in the climate of central Spain, which is supposed to have "nine months winter and three months hell."

Although the book is small, it contains a remarkable amount of valuable information not only in the mode of handling the wounded,

but also the equipment of mobile hospitals and the actual surgical treatment of war wounds. This has been done because all extraneous matter has been removed. Nothing but valuable facts have been printed. This is a feature which a good many of our medical books lack.

SURGERY OF MODERN WARFARE, Volume I, By 65 contributors, edited by Hamilton Bailey, F. R. C. S., surgeon, Royal Northern Hospital, London. The Williams and Wilkins Company, Baltimore, Md., publishers, 1941. Price, \$10.

Military surgeons, young and old, will obtain from this book a much better understanding of treatment of war wounds. The young surgeon gets a clear and concise description and technic of "debridement" and "wound excision" and the fundamental difference in each. After reading this chapter one will never make the mistake of wound excision when debridement is indicated or vice versa. The older surgeon will profit by the technic and description of the "closed method" of treatment (Orr-Truetta method) of wounds and compound fractures as compared to the Dakin methods so universally used in World War I.

To this reviewer Section 111, Wounds of the Trunk is the most interesting and stimulating in the whole volume. The Evolution of the Abdominal Surgery of War and Laparotomy for War Wounds are the most outstanding contributions. It not only tells the surgeon how and when but also why. In this chapter the author reviews the premise of the interventionist and the abstentionist and shows how high-explosive shells produced lesions which failed to respond to conservative measures. It was this period, after 1915, when the mortality was appalling that our present-day attitude, i. e., early operation, was achieved. Definite surgical procedures for the great multiplicity of lesions of the chest and abdomen as the result of wounds sustained in modern warfare are outlined and illustrated.

The chapters devoted to wounds of the blood vessels and nerves and tendons and methods of immobilization of limbs are clearly presented. The chapter, Wounds of Bones and Joints, is short but contains pertinent information in the space allowed. Technics are exact. General surgeons will find in these chapters handy references which will make them better military surgeons.

This book is a "must" for every surgeon entering the military service for it presents surgical problems in a light vastly different from that encountered in civil practice.

WAR WOUNDS AND INJURIES, edited by E. Fletcher, M. A., M. B., M. R. C. P. Physician, Emergency Medical Service; physician Queen Mary's Hospital for the East End; assistant physician, British Red Cross for Rheumatism, and (with physical medicine) Royal Free Hospital; and R. W. Raven, F. R. C. S., surgeon, Emergency Medical Service; surgeon to out-patients, French Hospital, London; assistant surgeon, Royal Cancer Hospital (Free) and Gordon Hospital;

late Arris and Gale lecturer and Erasmus Wilson lecturer, Royal College of Surgeons; with a foreword by Lord Horder. Pp. 262. A William Wood book. The Williams & Wilkins Co., Baltimore, Md., 1940. Price \$5

The authors are also the editors of the *Postgraduate Medical Journal* and it was their intention in writing this book to secure and correlate the modern views regarding the management of war wounds and injuries.

The book is divided into 19 chapters written by chosen contributors and each chapter treats on various war wounds and injuries in relation to the anatomical sites, such as wounds of the chest, head, eyes, etc. Each contributor is a specialist in his field and many gained experience during the great war of 1914-18. The book is small, could be used as a compend, and would be of value to anyone interested in this type of surgery.

MEDICAL DISEASES OF WAR by Sir Arthur Hurst, M. A., D. M., Oxon., F. R. C. P., Lieut.-Col., Late R. A. M. C.; consulting physician to Guy's Hospital, and late officer commanding Seale Hayne Hospital for functional nervous disorders; member of Medical Advisory Committee, Mediterranean Expeditionary Force, and consulting physician to the Salonica Army. With the cooperation of H. W. Barber, M. A., M. B., Cantab., F. R. C. P. physician-in-charge of the skin department, Guy's Hospital, F. A. Knott, M. D., Lond., M. R. C. P., bacteriologist to Guy's Hospital, and T. A. Ross, M. D., Edin., F. R. C. P., late medical director of Cassel Hospital for functional nervous disorders. 327 pages, Williams & Wilkins Co., Baltimore, publishers, 1940. Price \$5.50

This is a revision of a book first published in November 1916 as *Medical Diseases of the War* and now reprinted as *Medical Diseases of War*. The most striking feature to a medical officer is that half of the work is devoted to hysteria and anxiety neuroses, showing how strong is the unconscious defense against the hazards of war. Furthermore, this does not include the cardiac neuroses, soldier heart, and aged syndrome. If these are included, two-thirds of the book are given to functional disorders. The rest of the book is devoted to trench fever, the dysenteries, typhoid, tetanus, and some of the diseases frequent among troops. There is a good section on skin diseases. The volume is of convenient size and is a practicable little compend on the subject.

TOUGHEN UP, AMERICA! by Victor G. Heiser, M. D. Published by Whittlesey House, McGraw-Hill Book Co., Inc., New York, 1941. Price \$2.

Fourteen chapters and 228 pages, telling what we can do as individuals to "toughen up" and get the most out of life in years to come.

THE MASK OF SANITY by Hervey Cleckley, B. S., B. A. (Oxon.), M. D., professor of neuropsychiatry, University of Georgia School of Medicine, Augusta, Georgia. C. V. Mosby Co., St. Louis, Mo., publishers, 1941. Price \$3.

The author reinterprets the so-called psychopathic personality in that there is a mask of perfect sanity concealing the real mental state

of these people. This mask of sanity complicates the diagnoses, continually deceives the physician, the law, and the community. The book is well written, understandable, and the problem well presented.

INTRODUCTION TO PSYCHOBIOLOGY AND PSYCHIATRY, A Textbook for Nurses by *Esther Loring Richards, M. D., Sc. D., associate professor of psychiatry, Johns Hopkins University; physician-in-charge of the out-patient department, Henry Phipps Psychiatric Clinic, Johns Hopkins Hospital; psychiatrist-in-chief, Baltimore City Hospitals.* Pp. 357. The C. V. Mosby Co., St. Louis, Mo., publishers, 1941. Price \$2.50.

A well written, easily understandable book. The author gives a good insight in the study of functioning in normal behavior and discusses very clearly psychopathology. The book is a definite asset to a nurse's library.

HYGIENE, A Textbook for College Students on Physical and Mental Health from Personal and Public Aspects by *Florence L. Meredith, B. Sc., M. D., Fellow of American Medical Association, American Public Health Association, American Psychiatric Association, professor of Hygiene, Tufts College.* Foreword by *Frank Howard Lahey, M. D., D. Sc., LL. D., president-elect, American Medical Association.* Third edition, Pp. 822, illustrated. The Blakiston Co., Philadelphia, Pa., publishers, 1941. Price \$3.50.

The author starts with a dominating theme—what health situations exist in the life of individuals and peoples—and this theme continues throughout the 49 chapters. It gives a concise scientific action appropriate to the layman, college student, or nurse, but not in sufficient detail to be of greatest value to the practicing physician.

The chapter devoted to "The Health Situation in the United States" deals with the life span, statistical evidence, biological evidence, the cause of senility, the average length of life, the 10 leading causes of death, the decline in mortality from communicable diseases, and fatality rates in an unusually clear and practical manner and shows that while as a Nation we are "adding fitness and subtracting from sickness," there is much to be done before the statistically well become the biologically well.

The chapter on food is especially well written and contains much of the new knowledge on vitamins. The chapter on diet is a splendid summation of both the national and world situations and offers sound solutions to these problems.

The chapter on mental hygiene is well adapted to the medical social worker's peculiar needs and if carefully read, would be of considerable assistance to such workers in the present emergency.

The book is splendidly illustrated and well bound.

APPROVED LABORATORY TECHNIC, Clinical Pathological, Bacteriological, Mycological, Parasitological, Serological, Biochemical, and Histological. By *John A. Kolmer, M. S., M. D., Dr. P. H., Sc. D., LL. D., L. H. D., F. A. C. P., professor of medicine, Temple University; director of the research institute of cutaneous medicine, Philadelphia; formerly professor of pathology and bacteriology,*

Graduate School of Medicine, University of Pennsylvania, and Fred Boerner, V. M. D., assistant professor of bacteriology, School of Medicine and Graduate School of Medicine, University of Pennsylvania; bacteriologist, Graduate Hospital, Philadelphia. Third edition, 921 pages. D. Appleton-Century Co., New York, publishers, 1941. Price \$8.

The third edition of this widely used and well-known text within a period of three years from the appearance of the second edition is evidence of the endeavors of the authors to bring constantly changing and improved methods of laboratory technic before the medical profession. This new edition follows the same general plan as that of the previous one. The technic of the methods has been approved by the members of a group of 28 collaborators.

Almost all chapters have undergone revisions in varying degrees, some minor only, but all directed toward complete modernization. An appendix has been added in which are grouped a number of the newer and more important laboratory procedures which have been developed largely during the past 3 years.

The description of technic occurs in a detailed and concise manner. Sources of error are emphasized. The illustrations are quite adequate. There are included well-written chapters on histopathological technic and methods for toxicological examinations, chapters not always included in textbooks of this nature. The chapter on diagnostic bacteriological methods is excellent. A large part of it is devoted to methods of identification of almost all the important pathogenic micro-organisms, each one being considered separately.

Within the covers of the third edition of *Approved Laboratory Technic* can be found practically all tests that are worthwhile in the field of clinical pathology today. It can be recommended as a valuable and authoritative manual for all those working in the field of laboratory medicine.

THE PHARMACOLOGICAL BASIS OF THERAPEUTICS, A Textbook of Pharmacology, Toxicology, and Therapeutics for Physicians and Medical Students, by Louis Goodman, M. A., M. D., assistant professor of pharmacology and toxicology, Yale University, School of Medicine, and Alfred Gilman, Ph. D., assistant professor of pharmacology and toxicology, Yale University, School of Medicine. Pp. 1383. The Macmillan Co., New York, publishers, 1941. Price, \$12.50.

The authors have correlated the basic physiological principles of pharmacology and the actions and uses of drugs with pharmacodynamics and the pathological physiology of disease. Much emphasis is placed on the basic principles of drugs and on their therapeutic procedures in relation to their clinical applicability.

The book is divided into sections and each section is classified as to the physiological effect of drugs on the various systems of the body. One chapter is devoted to drugs of endocrine origin, such as the male and female sex hormones, and one chapter deals with the water and

fat soluble vitamins including vitamin K. The appendix is composed of 20 pages and is devoted to instruction in the principles of prescription writing. The index makes the material easily accessible as it is organized with the drugs grouped on a basis of therapeutic use, as well as a listing of the diseases. There are 1,383 pages, 129 figures, and 67 tables. The book is recommended for those interested in research, or for students and teachers of therapeutics.

While the book is rather technical for the general practitioner, he will find it a valuable reference of the latest information on the composition and effects of drugs.

THE PHARMACOLOGY OF ANESTHETIC DRUGS, A Syllabus for Students and Clinicians, by *John Adriani, M. D., instructor in anesthesia, New York University College of Medicine; assistant visiting anesthetist, Bellevue Hospital.* Second edition, 86 pages. Charles C. Thomas, Springfield, Ill., publishers, 1941. Price \$3.50.

This book is an outline of physiological and pathological changes occurring in various organs and systems during anaesthesia and acquaints the student anaesthetist "with pharmacological facts relating to drugs in current use." The author has divided his material into 13 chapters, each of which deals with a certain phase of anaesthesia or reaction of drugs, such as absorption and elimination, general systemic effects, disturbances of respiration, etc. The physiological and pathological changes are shown in diagrams which illustrate clearly the mechanics involved.

The bibliography is exceptional and alone is well worth the price of the book. The index is complete and permits easy reference to any drug or anaesthetic. The book is recommended, particularly to students and anaesthetists, but anyone interested in anesthesia will find it of great value either as a reference or as an outline for study.

TEXTBOOK OF BIOCHEMISTRY by *Benjamin Harrow, Ph. D., professor of chemistry, City College, College of the City of New York.* Second edition, 439 pages, 88 illustrations. W. B. Saunders Co., Philadelphia, Pa., publishers, 1940. Price \$3.75.

This book is a concise, sound, and authoritative presentation of the fundamentals and recent advances in biochemistry. It is well bound, written in clear English, accurately indexed, thoroughly up to date, and shows evidence of having undergone a painstaking revision. The references listed at the end of each chapter are especially valuable for those wishing to obtain more detailed information in any particular field. The author not only lists the references but also points out their virtues as well.

This book was written primarily for the undergraduate student in biochemistry, although anyone desiring a clear, concise, up-to-date review of the subject, will find this text one of the best available.

PHYSICAL MEDICINE. *The Employment of Physical Agents for Diagnosis and Therapy* by *Frank H. Krusen, M. D., F. A. C. P., associate professor of physical medicine, the Mayo Foundation, University of Minnesota; head of the section on physical therapy, the Mayo Clinic; member of the Council on Physical Therapy of the American Medical Association; past president of the American Congress of Physical Therapy; past president of The Academy of Physical Medicine.* Pp. 846 with 351 illustrations. W. B. Saunders Co., Philadelphia, Pa., publishers, 1941. Price \$10.

Krusen's books on physiotherapy may be said to have culminated in this work which is almost as large as an Osler's "Practice of Medicine." It is a very complete book with a history of physical therapy and complete sections on all fields of thermo-therapy, light therapy, electro-therapy, hydro-therapy, and all mechanical measures and devices used in the field of physical treatment of disease. Of special interest is the section devoted to the organization and plan of a hospital department of physical therapy, one on the teaching of physical medicine. It is an extremely valuable book and should be in the library of every hospital.

METHODS OF TREATMENT by *Logan Clendening, M. D., clinical professor of medicine, Medical Department of the University of Kansas; attending physician, University of Kansas Hospitals; and Edward H. Hashinger, A. B., M. D., clinical professor of medicine, Medical Department of the University of Kansas; attending physician, University of Kansas Hospitals; attending physician, St. Luke's Hospital, Kansas City, Mo.* Seventh edition, 997 pages, illustrated, C. V. Mosby Co., St. Louis, publishers, 1940. Price \$10.

This well-known work on medical treatment requires no introduction to its many readers. The publication of a seventh edition is definite evidence of the value and popularity of this work. Dr. Hashinger has collaborated with Dr. Clendening in the revision of the volume for this edition.

The general plan of the book has been retained although in many places the book is entirely recast, and entirely new sections or chapters have been included. Among these are: chemotherapy for coccal diseases, including the use of sulfanilamide and its derivatives; back-ache; peripheral vascular diseases; Raynaud's disease and scleroderma; deficiency diseases; anesthetics; and gout. The sections on intestinal parasites, syphilis, digitalis, extracts of ductless glands, vitamins, physical therapy, transfusion, radiotherapy, pneumonia, allergy, cardiac failure, diseases of the ductless glands, and peptic ulcer have been completely rewritten. Many new drugs such as heparin, benzedrine, aluminum hydroxide, and dilantin are discussed. Dr. Wood and Dr. Knight have contributed original articles on artificial pneumothorax and diseases of the esophagus.

It is to be regretted that there is not a more complete discussion of the treatment of coronary thrombosis and acute and chronic cor-pulmonale. The description of the method of detecting pulses alter-

nans with the blood pressure apparatus is considered incomplete. We found no mention of the value of blood transfusion in the treatment of nephrosis, nor of the use of the Sulfonamide compounds in the treatment of lymphogranuloma venerum.

Regardless of these minor criticisms, however, this book is considered to be an outstanding work on medical treatment and is recommended as a valuable supplement to the library of all physicians.

THE EXTRA-OCULAR MUSCLES, A Clinical Study of Normal and Abnormal Ocular Motility, by *Luther C. Peter, A. M., M. D., Sc. D., LL. D., professor emeritus of diseases of the eye in the graduate school of medicine of the University of Pennsylvania; member of consulting staff in the graduate hospital of the University of Pennsylvania; consulting ophthalmologist in the Rush Hospital for Consumption and Allied Diseases, the Friends' Hospital for Nervous and Mental Diseases and the Roxborough Memorial Hospital; former member and president of the American Board of Ophthalmology; fellow and past-president of the American Academy of Ophthalmology and Otolaryngology; fellow of the American Ophthalmological Society, the American Medical Association, the American College of Surgeons, the College of Physicians of Philadelphia, the Oxford Ophthalmological Congress, etc.* Third edition, thoroughly revised, 368 pages, illustrated. Lea & Febiger, Philadelphia, publishers, 1941. Price \$4.50.

This is the third edition of *The Extraocular Muscles* which is used as a text on ocular motility by most ophthalmologists.

The text is divided into 5 parts arranged in logical sequence of normal and abnormal ocular motility. Part 1 deals with the anatomy and physiology of the ocular muscles. Part 2 deals with the diagnosis and treatment of heterophoria. Part 3 deals with the diagnosis and treatment of heterotropia. In this part there is considerable discussion dealing with orthoptic training in the development of fusion. This very essential topic was not included in previous editions. The various instruments such as the synoptophore, cheiroscope, and stereoscope are discussed and evaluated as to their proper scope in fusional training. It is of interest to note the author states that in concomitant squint of moderate degree in children under seven, 50 percent can be cured without operation by improved technic developed in the past decade. Part 4 discusses paralytic squint. Chapter 5 was written in collaboration with Dr. Joseph C. Yaskin, Professor of Neurology in the graduate school of Medicine, University of Pennsylvania. The distinguishing features of vestibular ocular and neurologic nystagmus are clearly outlined. Chapter 6 discusses surgical technic, instruments required and the technic for the standard muscle operations of recession, resection, tucking, and advancement.

GONORRHEA IN THE MALE AND FEMALE, a Book for Practitioners, by *P. S. Pelouze, M. D., assistant professor of urology, University of Pennsylvania; consulting urologist to Delaware County Hospital; special consultant to United*

States Public Health Service; member of board of directors, American Social Hygiene Association and American Neisserian Medical Society. Third edition reprinted, with new chapters on sulfanilamide and its derivatives and on gonorrhea in immature females. Pp. 489. W. B. Saunders Co., Philadelphia, publishers, 1941. Price, \$6.

This book has been on the Navy Standard Book list for several years and still holds the distinction of being the one complete modern text on the subject.

In this third edition the book is further modernized, particularly as to the newer forms of treatment.

Eleven new chapters have been added on such phases as social hygiene, various public-health aspects, clinic management, etc. In these times when venereal-disease control is of extreme importance among our armed forces, every medical officer would be well repaid by a reading of the public-health section of the book.

Happily, the new chapters and modernizing process have not enlarged the book, so that it is just as valuable as ever to the person who undertakes to treat the gonorrheal individual or who is interested in gonorrhea as a broad public-health problem.

THE FOOT AND ANKLE, Their Injuries, Diseases, Deformities, and Disabilities, by Philip Lewin, M. D., F. A. C. S., *associate professor of bone and joint surgery, Northwestern University Medical School; professor of orthopedic surgery, Postgraduate Medical School of Cook County Hospital; attending orthopedic surgeon, Cook County Hospital; attending orthopedic surgeon, Michael Reese Hospital, Chicago; consulting orthopedic surgeon, Municipal Contagious Hospital, Chicago.* Lea & Febiger, Philadelphia, Penna., publishers, 1940. Pp. 620, 303 illustrations. Price \$9.

The author has dedicated this book to Dr. Allen B. Kanavel who once suggested that a book be written on the foot, covering that subject as Dr. Kanavel had done for the hand. Dr. Lewin has accomplished much toward that goal.

In the past few years, there have been many excellent books on disorders of the foot. This work has included many excellent features of those textbooks, and in addition, the author has enlarged the scope of his text to include the ankle.

Static and mechanical conditions of the foot and ankle, mechanics of the physiology of foot action, etiology and symptoms of foot strain, all are excellently discussed with all the accepted methods of treatment. It is particularly practical in the descriptions of treatment of minor foot conditions and well adapted for use by the general practitioner in this respect.

The foot and ankle discussed in relation to all the other specialities, is an excellent feature of the book.

The book is well illustrated and has excellent and adequate descriptions of operation procedures. It deserves a place on the desk

of the general and orthopedic surgeon as well as the general practitioner.

UROLOGICAL NURSING by *David M. Davis, M. D., associate in urology, The Johns Hopkins University, 1917-24; assistant professor of urological surgery, University of Rochester, and urologist, Strong Memorial Hospital, 1924-28; assistant visiting urologist and dispensary urologist, The Johns Hopkins Hospital, 1928-30; visiting urologist, Desert Sanitarium, Tucson, 1931-35; professor of urology, Jefferson Medical College, Philadelphia, 1935.* Third edition, revised, illustrated. Pp. 203. W. B. Saunders Co., Philadelphia, publishers, 1941. Price \$2.25.

The author has revised this book for nurses and gives the most recent knowledge about urological diseases. After describing concisely, yet clearly, the anatomy and physiology of the urogenital tract and giving illustrations, he mentions diseases and symptoms, contributing a chapter to urinary obstructions and principles of drainage. This is most essential. Various drugs used in the treatment of urinary infections are listed giving their importance and reaction. Sulfathiazole at present is preferred.

More time is taken to explain the nursing care, both preoperative and postoperative and urological procedures giving the latest technic and treatment. Dr. Davis' aim is to have urological nursing better understood so that it will have its place with other branches of medicine and surgery. Formerly it did not.

The chapter describing and picturing instruments, their usage and care, is highly rectified. Without modern well-protected equipment, the surgeon is hindered and the patient suffers.

A nomenclature concludes the textbook which is complete and illuminating, yet tersely compiled.

BRUCELLOSIS (Undulant Fever) Clinical and Subclinical by *Harold J. Harris, M. D. Health Officer, Westport, N. Y.; consulting physician, St. Lawrence State Hospital; attending physician, Elizabethtown Community Hospital; Lieutenant Commander, Medical Corps, United States Naval Reserve; member, New York Academy of Medicine; associate member, American College of Physicians.* Foreword by *Walter M. Simpson, M. D., F. A. C. P., director, Kettering Institute for Medical Research, Miami Valley Hospital, Dayton, Ohio.* Pp. 286, with 12 colored and 44 black and white illustrations. Paul B. Hoeber, Inc., New York, publishers, 1941. Price \$5.50.

This is a fine monograph on an important disease which has presented many perplexing problems to the clinician and to the epidemiologist. The book starts off well with a fine picture of Sir David Bruce, whose work on the etiology of brucellosis, or as it was first called, "Malta fever," is one of the classics of medical history. The etiology, pathology, symptomatology, and treatment are given in detail. An important feature and one in which the clinician should be particularly interested is the very careful discussion and the great amount of space given to the mild types of the disease—often overlooked and given other

diagnoses. There is a fine bibliography and a good index. The printing and binding can scarcely be surpassed.

CEREBROSPINAL FEVER by *Denis Brinton, D. M. (Oxon), F. R. C. P. (Lond.)*, physician in charge of the department for nervous diseases, St. Mary's Hospital, London; assistant physician to out-patients, National Hospital for Nervous Diseases, Queen Square, London; physician to the Royal London Ophthalmic (Moorfields) Hospital, London, consultant neurologist to the London County Council. Pp. 163. The Williams & Wilkins Co., Baltimore, Md., publishers, 1941. Price \$3.

Much of this monograph is devoted to the historical, bacteriological, and clinical aspects of the disease but brings out nothing particularly new in these matters. The author's concept of cerebrospinal fever is that it is a nasopharyngeal disease in which the further development of septicemia and meningitis occurs in a relatively few susceptibles. The original mortality of from 70 to 90 percent has been reduced to 33 percent by serum therapy.

In England, due to overcrowding in shelters in January, February, and March of 1940, there were half as many cases as during the entire four years of the World War. The season (winter), the age factor (young people), and overcrowding with poor ventilation are the epidemiological factors responsible for epidemics. The search for carriers is a waste of time.

Treatment with sulphonamides has reduced the mortality in 4,388 cases to 23.7 percent in civilians and to 11.3 percent in noncivilians with rates as low as 6 to 7 percent (Perry) in a series of 900 Army cases; no serum has been used.

In order of preference one may use (1) sulfapyridine, (2) sulfanilamide, and (3) sulfathiazole, the latter probably being as satisfactory as any. The concentration of the drug should be kept at from 1 to 2 mgm. per 100 cc. of spinal fluid, and well below 5 mgm. The dose for adults should be for the first two days, 9½ grams per 24 hours divided into equal doses every four hours. For the next 2 days, 6 grams, and the next two days 3 grams; in all, 36 to 50 grams.

The effect is striking, and if given early may be expected to effect a relief of symptoms promptly and to result in a cure in most cases, hence the importance of an early bacteriological diagnosis of the disease. With proper diagnosis, treatment with sulphonamides, and control of ventilation, the disease can be readily controlled.

DISEASES OF THE DIGESTIVE SYSTEM, edited by *Sidney A. Portis, B. S., M. D., F. A. C. P.*, associate clinical professor of medicine, Rush Medical College of the University of Chicago; attending physician, Michael Reese Hospital; consulting physician, Cook County Hospital, Chicago. Pp. 962, illus. Lea & Febiger, Philadelphia, publishers, 1941. Price \$10.

This textbook on diseases of the digestive system presents in one volume articles contributed by 50 physicians, each of whom has made

a special study in the branch of gastroenterology of which he writes. As the editor states in the preface, he has attempted to select contributors "whose clinical experience is broad enough to justify a contribution in their subject."

The book is divided into 5 parts. The first introductory part is made up of chapters on the history of diseases of the gastrointestinal tract by Major, the anatomy of the gastrointestinal tract by De Garis, the physiology by Necheles, and the interpretation of gastrointestinal tract pain by Chester M. Jones. Part 2 covers the etiologic factors concerned in gastrointestinal disease, including the gastrointestinal manifestations of cardiovascular, urogenital, metabolic, endocrinologic, and allergic conditions, together with a discussion of the metazoan parasites of the gastrointestinal tract and chapters on the neuroses and neurogenic disturbances.

Part 3 is concerned with diseases of the esophagus, stomach, and duodenum and includes a chapter on gastritis by Schindler with observations on gastroscopy. Part 4 includes diseases of the liver, gall bladder, pancreas, and small intestine, with a chapter on regional ileitis by Crohn and a discussion of the vermiform appendix by Wangersteen. Part 5 takes up the diseases of the large bowel and rectum, including functional diseases of the colon, protozoal infections, bacillary dysentery, nonspecific ulcerative colitis, "mucous colitis," cancer, diverticulosis, and diverticulitis, with concluding articles on melanosis coli, hemorrhoids, and lymphopathia venereum by Dudley Smith and Sipman.

It is considered that Dr. Portis has been admirably successful in compiling this complete and highly authoritative work and that this book is one which few physicians can afford to be without.

TEXTBOOK OF PEDIATRICS by J. P. Crozer Griffith, M. D., Ph. D., *emeritus professor of pediatrics in the University of Pennsylvania; consulting physician to the Children's Hospital, Philadelphia; consulting physician to St. Christopher's Hospital for Children; consulting pediatricist to the Woman's the Jewish, and the Misericordia Hospitals, etc; corresponding member of the Societe de Pediatrie de Paris, and A. Graeme Mitchell, M. D., B. K. Rachford professor of pediatrics, College of Medicine, University of Cincinnati; medical director and chief of staff of the Children's Hospital of Cincinnati; director of the Children's Hospital Research Foundation; director of pediatric and contagious services in the Cincinnati General Hospital.* Third edition, revised and reset; 991 pages, illustrated. W. B. Saunders Co., Philadelphia, Penna., publishers, 1941. Price \$10.

This third edition has been completely revised and rewritten. The contributors have done special work in the field of pediatrics and are well-known specialists. The style and arrangement of the chapters have been kept uniform, as well as to their size. The chapters on mental growth and development have been added besides a number of new illustrations, charts, tables, and figures. There are 12 sections

and each section is classified according to the diseases in relation to the body systems, or to finding, growth, and development, etc. There are 34 pages of excellent index. The book is recommended for the student, general practitioner, and pediatrician.

A DIABETIC MANUAL, for the Mutual Use of Doctor and Patient, by *Elliott P. Joslin, M. D., Sc. D., clinical professor of medicine emeritus, Harvard Medical School; medical director, George F. Baker Clinic at the New England Deaconess Hospital; consulting physician, Boston City Hospital, Boston, Mass.* Seventh edition, thoroughly revised, 238 pages, illustrated. Lea & Febiger, Philadelphia, publishers, 1941. Price \$2.

This is the seventh edition of this well-known book. The author, well known in his field, has divided his book into 29 chapters and each chapter deals separately with some phase of the diagnoses, treatment, or complications of the diabetic. He tells clearly the technic of giving insulin and warns of its dangers. Several chapters are devoted to diets, weights, and laboratory diagnosis and one chapter deals with food and its composition. The book is small and contains 238 pages. It is well illustrated with photographs, diagrams, and tables.

ARTHRITIS and Allied Conditions by *Bernard I. Comroe, A. B., M. D., F. A. C. P., instructor in medicine, University of Pennsylvania; senior ward physician, Hospital of the University of Pennsylvania.* Second edition, 878 pages, illustrated with 242 engravings. Lea & Febiger, Philadelphia, Penna., publishers, 1941. Price \$9.

This is a second edition made necessary both by the popularity of the first edition and the new ideas of treatment which have been advanced. These latter include the use of the sulphonamides, gold, home methods in physical therapy, new evidence in regard to the value of diathermy, hydrotherapy, vaccines, chaulmoogra oil, histamine, sulphur, bee venom, and a number of others. Home management and the general practitioner's problem are stressed. An interesting feature is a list of American spas with approximate prices to the patient. A valuable book for both internist and general practitioner particularly.

NATURAL RESISTANCE AND CLINICAL MEDICINE by *David Perla, B. S., M. D., late pathologist and bacteriologist, Montefiore Hospital, N. Y., instructor in medicine, Columbia University College of Physicians and Surgeons; and Jessie Marmorston, B. S., M. D., formerly bacteriologist to Montefiore Hospital; assistant in pathology, Cornell University, Medical College.* pp. 1344. Little, Brown & Co., Boston, Mass. publishers, 1941. Price \$10.

This book presents a well written and arranged volume dealing very completely with our present facts and theories of body resistance.

The large volume is divided into 10 sections, dealing with every phase of the subject as follows: heredity, age, sex, endocrine glands, humoral and cellular mechanisms, the role of the liver, body surfaces

and nervous system, diet, including the vitamins, climate and clinical aspects of resistance. The sections dealing with the endocrine glands and diet, including the vitamins are particularly interesting and instructive.

Each chapter is followed by a concise summary, and an extensive bibliography. The index at the close of the volume is especially complete and convenient.

This book will be found invaluable as collateral reading for the student of medicine on the subject of body resistance to disease.

ANUS, RECTUM AND SIGMOID COLON, Diagnosis and Treatment, by *Harry Ellicott Bacon, B. S., M. D., F. A. C. S., F. A. P. S.*, *clinical professor of proctology, Temple University School of Medicine; associate professor of proctology, Graduate School of Medicine, University of Pennsylvania; visiting proctologist, St. Luke's and Children's Hospital; proctologist, National Stomach Hospital; consultant proctologist, Mercy Hospital; consultant proctologist, Paul Kimball Hospital, Lakewood, N. J.; co-founder and past president, Proctologic Society Graduate Hospital, University of Pennsylvania; Lieutenant-Commander, MC-V (Surg.), United States Naval Reserve.* Second edition, pp. 857, illustrated. J. B. Lippincott Co., Philadelphia, publishers, 1941. Price \$8.50.

This practical and complete work on the lower bowel is truly an encyclopedic type of book. However, the subject matter is by no means merely a recapitulation of the experience and methods of others.

The author has been a most energetic and intensive worker in close association with some of the world's foremost authorities on the subject of proctology. The fruits of this diligent effort and priceless association combined with wide clinical experience have been embodied in the text of his book.

The chapter dealing with "Lymphopathia Venereum" constitutes the most comprehensive discussion of this subject to be found, I believe, in any textbook.

Notable also is Doctor Bacon's discussion of endometrioma or "endometriosis."

The chapter dealing with malignancy of the anus, rectum, and sigmoid colon—a subject of ever increasing and worthy interest—contains a very complete and excellent digest of the most modern and generally recognized surgical procedures applicable in various cases and is fully amplified by numerous clear illustrations.

The author's liberal discussion of pilonidal cysts and their treatment, particularly as pertains to the open versus the closed method, should prove most interesting and enlightening to military surgeons who appreciate the real problem often presented by this condition.

The use of chart forms as a method of listing the subject matter, in certain instances, such as the tabulation of differential diagnosis in related conditions, affords the reader an easy and expeditious means of gaining information. Likewise is the summary of treatment at the completion of most chapters distinctly helpful.

This book is clearly printed in easily legible type, excellently illustrated and well-bound; and aside from being a rich compendium of attractively and painstakingly recorded information must arouse in the minds of its readers an appreciation of the scope and importance of the subject with which it deals and should contribute very definitely towards fixing proctology in the place it deserves alongside of the other major specialities of medicine and surgery.

THE TREATMENT OF FRACTURES by *Dr. Lorenz Bohler, director of the Hospital for Accidents, Vienna; lecturer on surgery in the University of Vienna.* Fourth English edition, translated from the fourth enlarged and revised German edition by *Ernest W. Hey Groves, M. S., M. D., F. R. C. S., consulting surgeon, Bristol General Hospital; emeritus professor of surgery, University of Bristol; late examiner in surgery, Universities of London, Liverpool, Leeds, Manchester, Sheffield, and the National University of Ireland.* With 1059 illustrations. A William Wood book. Williams & Wilkins Co., Baltimore, Md., publishers, 1941. Price \$10

This is not a new edition but simply a reprint of a popular text. The new edition was in the process of preparation when war was declared and it probably will not be revised until after the war. The book, however, is still valuable as the principles laid down by the author in treating of fractures are still used by those specializing in this branch of medicine.

FIRST AID IN EMERGENCIES by *Eldridge L. Eliason, A. B., M. D., Sc. D., F. A. C. S., professor of surgery, University of Pennsylvania School of Medicine; professor of Surgery, University of Pennsylvania Graduate School of Medicine; surgeon, University of Pennsylvania, Presbyterian, and Philadelphia General Hospitals.* Tenth edition completely revised and reset, 126 illustrations. J. B. Lippincott Co., Philadelphia, publishers, 1941. Price \$1.75

An excellent little book for the lay person interested in first aid. It is recommended for teaching or to accompany a first-aid kit.

EMERGENCY SURGERY by *Hamilton Bailey, F. R. C. S. (Eng.) surgeon, Royal Northern Hospital, London; surgeon and urologist, Essex County Council; surgeon, Italian Hospital; consulting surgeon, Clacton Hospital and the County Hospital, Chatham; external examiner in surgery, University of Bristol.* Fourth edition, with 930 illustrations. Williams & Wilkins Co., Baltimore, Md., publishers, 1941. Price \$15

The theme of this work is, in the words of the author, "When to operate, when not to operate, and how to operate under emergency conditions." It was written essentially for the isolated surgeon and the occasional operator, and not necessarily for the specialist in surgery.

The subject of emergency surgery must necessarily be the concern of every physician, and the need for a comprehensive, up-to-date and ready reference is at once apparent. The task of adequately covering a subject of such wide scope, and limiting the work to a single volume has been admirably achieved. The book has been kept abreast of the

times by frequent editions, this being the fourth edition in 10 years.

The text is written in a clear and concise manner. Personal experiences of the author are freely described and many brief case reports are given. Essential points in diagnosis, treatment, and operative technic are covered briefly but adequately, and to each chapter is appended a list of references for those wishing information in great detail.

The book is richly illustrated throughout, there being 930 illustrations, of which many are in color. This does much to clarify practical points in diagnosis and operative technic and adds greatly to the value of the book as a guide to any physician who may be called upon to treat a surgical emergency.

This book is highly recommended as a valuable addition to the personal library of any physician or surgeon.

ABDOMINAL OPERATIONS by Rodney Maingot, F. R. C. S. Eng., senior surgeon to the Southend General Hospital, and to the Royal Waterloo Hospital, London; consulting surgeon to the Southend Municipal Hospital, and to the Runwell Hospital; surgeon, Emergency Medical Service; Fellow of the Association of Surgeons of Great Britain and Ireland; late chief assistant to a surgical unit, St. Bartholomew's Hospital, London. Volumes I and II, pp. 1385, illustrated. D. Appleton-Century Co., New York, publishers, 1940. Price \$18

This two-volume, first-edition, treatise on abdominal operations is a welcome addition to the medical library. The author not only gives a detailed consideration of the modern technic, but also deals with the choice of operation in each particular case and the difficulties which sometimes arise during operation.

The 2 volumes are divided into 11 parts, 5 of which are covered in the first volume and 6 in the second volume.

The first part deals with abdominal incisions. The author describes the various types of abdominal incisions discussing the advantages and disadvantages of each. Also in this part is included the management of the wound and wound complications. One part of the book deals with the stomach and duodenum. The author not only describes the surgical treatment, but takes up the pathology and symptoms of the various surgical conditions. The different operative procedures of the stomach and duodenum are described, and an interesting feature of the book throughout, particularly for the young surgeon, is that, after discussing and describing the various operations, the author recommends the operation which he has found to be best suited for each condition. Part of the first volume is also devoted to the surgical aspects of the spleen, pancreas, the gall bladder, and bile ducts.

In the second volume parts are devoted to the liver, appendix, peritoneum, mesentery, and the omentum. Hernias are discussed, including inguinal, femoral, umbilical, ventral, and strangulated external hernia.

The intestines, including injuries, tumors, regional enteritis, obstruction, and diverticuli are discussed in great detail. The last part deals with postoperative chest complications.

The author describes 4 clinical types of chronic appendicitis: (1) Recurrent appendicitis; (2) recurrent appendicular colic; (3) the grumbling appendix; and (4) appendix dyspepsia. This is quite interesting, as there is a question in the minds of some surgeons as to whether or not chronic appendicitis might not be a myth.

The books are well written, easy to read, and well bound. The printing is of large type on slick paper. The references are included directly in the body of the subject matter and are adequate. The illustrations are numerous and well done, making it possible to visualize the various operative procedures as described by the author in the text.

These excellent two volumes are highly recommended to the abdominal surgeons, resident surgeons, and to the medical officers in the various armed forces.

ESSENTIALS OF DERMATOLOGY by *Norman Tobias, M. D., senior instructor in dermatology, St. Louis University; assistant dermatologist, Firmin Desloge and St. Mary's Hospitals; visiting dermatologist, St. Louis City Sanitarium and Isolation Hospital.* Pp. 497, illustrated. J. B. Lippincott Co., Philadelphia, Pa., publishers, 1941. Price \$4.75

This is a very complete and tidy volume covering the entire field of dermatology. It is remarkable that so much detailed information can be compressed into so small a book. The description of all diseases and conditions with treatment is fully stated in a concise manner. There are many excellent and clear black and white illustrations. The book is well arranged, and the table of contents and index give ready reference. The book should prove a most useful and handy volume for medical students, practitioners, and those "not inclined to refer to larger standard dermatological text books," to whom it is highly recommended.

CLINICAL PELLAGRA by *Seale Harris, M. D., professor emeritus of medicine, University of Alabama, Birmingham, Ala., assisted by Seale Harris, Jr., M. D., formerly assistant professor of medicine, Vanderbilt University, Birmingham, Ala., with foreword by E. V. McCollum, Ph. D., Sc. D., LL. D., professor of biochemistry, School of Hygiene and Public Health, The Johns Hopkins University, Baltimore, Md.* Pp. 494, illustrated. The C. V. Mosby Co., St. Louis, Mo., publishers, 1941. Price \$7

In this book the reader will find the fascinating story of pellagra presented in a most readable manner. Without lengthy discussions the author gives a remarkably complete summary of the up-to-date knowledge of pellagra and of the many investigations which led to this knowledge. On controversial points he has an open mind, presenting generously the views of all. Perhaps some theories which recent discoveries have shown to be erroneous and which have no historical value could have been omitted.

The book is timely because, as the author notes, no other book on pellagra has been published in the last 2 decades. It is particularly well printed and amply illustrated.

DERMATOLOGIC ALLERGY, An Introduction in the Form of a Series of Lectures, by *Marion B. Sulzberger, M. D.*, assistant clinical professor of dermatology and syphilology, Skin and Cancer Unit of the New York Post Graduate Medical School and Hospital of Columbia University; associate attending in dermatology and syphilology, Montefiore Hospital, New York, N. Y.; consultant in dermatology and syphilology, French Hospital, New York, N. Y. Charles C. Thomas, Springfield, Ill., publisher, 1940. Price, \$8.50.

Entirely true to its subtitle, this 500-page monograph on dermatologic allergy is an introduction, albeit a thorough and complete one, to the subject. It is also a reproduction of a series of lectures, but this feature does not detract in the least (as in the case of some lecture-collection books) from its readability or utility as a text.

While it can indeed serve as an introduction for those students who contemplate a further and more exhaustive pursuit of the subject, the introduction is laid out in so comprehensive and so profound a form that any reader—we hope many a reader—will find here a welcome clarification of what was formerly a maze of confusion for him throughout the whole field of allergy.

The broad principles of the study are so ably and adequately expounded here that the reader is soon impressed with the scientific precision and possibilities of this rather new branch of medicine, especially if he is one of the many who have regarded it as a vague and groping endeavor.

The author's postulates as to the proofs of the existence of allergy as the causative basis in any given case are so rigid and so well worded as to remind one of Koch's Postulates.

The methods of diagnosis are so ramifying, so clearly outlined, contain so many elements of the Sherlock Holmes type of probing and lead the allergy worker into such bizarre paths that the reader is apt to become intrigued, not to say fascinated, by the whole lore of the thing.

Dr. Sulzberger's monograph is a contribution of unquestionable value to the literature of allergy and happily has brought his estimable lectures to an armchair audience which is much wider than the privileged ones of his lecture room.

DERMATOLOGIC THERAPY IN GENERAL PRACTICE, by *Marion B. Sulzberger, M. D.*, assistant clinical professor of dermatology and syphilology, Skin and Cancer Unit of the New York Post Graduate Medical School and Hospital of Columbia University; associate attending dermatologist, Montefiore Hospital, New York City, and *Jack Wolf, M. D.*, attending dermatologist and syphilologist, Skin and Cancer Unit of the New York Post Graduate Medical School and Hospital of Columbia University; director of dermatology, New York City Cancer Institute.

Pp. 680, illustrated. The Year Book Publishers, Inc., Chicago, Ill., publishers, 1941. Price, \$4.50.

This book, written from the standpoint of the general practitioner, stresses the therapeutic side of the more common dermatoses.

Clinical description, etiology, and prognosis are presented very briefly. This feature is believed to be quite advantageous in such a work. Differential diagnosis is briefly discussed, although some dermatoses could be omitted, such as the "distinctive chronic discoid, lichenoid, and exudative dermatosis of Sulzberger and Garbe."

The book is well organized with regard to material and is well indexed. In addition there is a short index of prescriptions for common skin conditions. There are 65 illustrations and the photographs are grouped for the purpose of comparison.

Syphilis and its therapeutics are included. This section uses about 75 pages and covers the subject briefly and well. There are seven tables giving treatment schedules. These are practically standard. The only objection to these tables is that rest periods are advised during the first year of treatment in late latent syphilis, "benign late syphilis," and late congenital syphilis. These rest periods vary from 2 to 3 weeks during the first year to as much as 6 weeks at the end of 18 months in some cases.

On the whole, this book justifies its title and existence. It is a handy therapeutic guide for quick reference, with sufficient bibliography for those who wish to delve further into the subject matter.

BACILLARY AND RICKETTSIAL INFECTIONS, Acute and Chronic, A Textbook, by William H. Holmes, professor of medicine, Northwestern University Medical School, chairman, department of medicine, Passavant Memorial Hospital, Chicago, Illinois. Pp. 676. The MacMillan Co., New York, publishers, 1940. Price, \$8.

Of the many subjects treated it would be difficult to select particular ones for special comment. Both the grouping of subject matter and the manner of presentation are unusual but logical, pleasing, and effective. The author has been unusually successful in his presentation of the historical, epidemiological, and preventive aspects of his subjects. One may take issue with him from time to time on isolated statements made or details presented, and indeed feel that it would have been better had they been omitted, yet on the whole one cannot fail to be impressed with the general value of the work. It is a real contribution to contemporary medical literature and could well set the pattern for other authors to follow. Unlike the standard text, this book can be placed by the reader's bedside, to be taken up at odd times to be read just as an interesting narrative would be. This increases its value both to the medical student and to the busy practitioner.

The reserve medical officer coming from civil practice to active service in the Navy will find the volume particularly valuable as a ready

means of orientation in such matters as plague, cholera, typhus, and other epidemiologic and clinical entities with which he is not apt to be particularly familiar unless he has had the advantage of previous training at the Naval Medical School.

The volume has still another and perhaps even greater value, in that it presents in a permanent and practical way the author's very valuable contribution to methodology in the field of medical education, particularly as it relates to the teaching of clinical medicine.

The book is attractively bound, the paper quite well chosen, and the type reasonably well selected, considering the fact that there is such a considerable amount of information presented in the 676 pages comprising the volume. Bibliographical references are numerous and well selected.

APPLIED ORTHODONTICS, by *James David McCoy, M. S., D. D. S., F. A. C. D.*, associate clinical professor of surgery (oral) School of Medicine, University of Southern California, Los Angeles, California; formerly professor of orthodontics in the College of Dentistry, University of Southern California, Los Angeles, California. Fifth edition, thoroughly revised. Pp. 333, illustrated with 227 engravings and a plate. Lea & Febiger, Philadelphia, publishers, 1941. Price \$4.50

This book presents a comprehensive treatise without verbosity of the science of orthodontics. The subject matter is logically presented in 333 pages of 20 sequent chapters, aptly illustrated with 227 engravings. Drawings not appearing in the four previous editions amplify the text.

The author stresses the necessity for case records and progress charts made available by gnathostatics, photostatics, the facial mask and the roentgenographic cephalometer. Such methods are invaluable guides for proper treatment as well as important instrumentalities in demonstrating clinical results.

This edition includes modern orthodontic procedures, related biologic considerations, with emphasis upon the skill of the orthodontist, rather than upon the use of certain treatment appliances. To those interested in the science of applied orthodontics this book will serve as a good text and handy reference.

EXODONTIA, by *M. Hillel Feldman, D. D. S.*, director of dentistry, Lincoln Hospital, Department of Hospitals, City of New York. Third edition, enlarged and thoroughly revised. Pp. 280, illustrated with 217 engravings. Lea & Febiger, Philadelphia, Pennsylvania, publishers, 1941. Price \$4

This work is well suited as a textbook for the dental student as it presents all the fundamental and elementary facts in this field in an excellent manner. This edition has been thoroughly revised and many of the later developments in exodontia have been included in it. Engraving and typography of the book are excellent.

THE DIAGNOSIS AND TREATMENT OF CARDIOVASCULAR DISEASE, edited by *William D. Stroud, B. S., M. D., F. A. C. P.*, professor of cardiology, University of Pennsylvania Graduate School of Medicine; president of the American Heart Association; treasurer and member of Board of Regents, American College of Physicians; licentiate of the National Board of Medical Examiners and the American Board of Internal Medicine; cardiologist to the Pennsylvania Graduate, Bryn Mawr, Abington Memorial, St. Christopher's and Children's Heart Hospitals. 56 contributors; fully illustrated; 2 volumes. F. A. Davis Co., Philadelphia, publishers, 1940. Price \$18 a set

This treatise on cardiology consists of two volumes that have been compiled by 56 outstanding contributors and excellently edited by William D. Stroud.

The binding is exceptionally good and the print such that reading is easy. Both volumes are well illustrated by drawings, charts, and photostats. Together the two volumes present the subject of cardiovascular diseases in a wider and more detailed manner than has been done before.

In reality it is a most complete symposium on the entire subject of cardiovascular diseases and the allied diseases, written by the outstanding men in their respective fields.

In reading these two volumes one is struck with the idea that he is reading a most interesting series of articles in a medical journal with its many illustrations and case histories, each chapter having an entirely different style and having its own list of references.

Probably the only just criticism is the arrangement of the chapters in the first volume, it would seem better to have the chapters on the normal heart and electrocardiography at the beginning rather than at the end of the volume.

There are only two or three chapters that are not interesting while there are many that are outstanding. The following are deserving of special mention.

1. Electrocardiography: By Samuel Bellet, M. D., and Thomas M. McMillan, M. D.
2. Rheumatic heart disease: By Currier McEwen, M. D.
3. Congestive Heart Failure: By Albert W. Bromer, M. D., and William A. Stroud, M. D.

This treatise can be recommended to the under-graduate, the practising physician, and to the cardiologist for its scope is wide enough to be of interest and instruction to all.

HEART FAILURE by *Arthur M. Fishberg, M. D.*, associate in medicine, Mount Sinai Hospital, New York City. Second edition, thoroughly revised. 829 pages, illustrated with 25 engravings. Lea & Febiger, Philadelphia, publishers, 1940. Price \$8.50.

The second edition of this well-known book will be welcomed by all physicians interested in the mechanism and treatment of circulatory failure as a highly authoritative source of reference.

The incorporation of advances made in this field since the publication of the first edition has resulted in changes in all the chapters as well as the addition of new material.

Essentially, the book is divided into three parts. The first part describes the individual manifestations of circulatory failure, the concept and types of failure including Harrison's classification of forward and backward failure together with Wollheim's classification on the basis of blood volume. The second part discusses the clinical picture resulting from circulatory failure, while the third part takes up the treatment of circulatory failure. Of interest to the surgeon as well as to the internist is the discussion of the mechanism, clinical picture and treatment of peripheral circulatory failure and shock.

The practical application of various technical procedures for determining the circulation time, venous pressure, and cardiac output is clearly set forth. The mechanism, clinical significance, and treatment of such manifestations as orthopnoea, nocturnal dyspnoea, pulmonary edema, Cheyne-Stokes breathing, pulsus alternans, and gallop rhythm are thoroughly discussed. The excellent bibliography has been brought up to date.

This book is recommended, not only for those who have a particular interest in cardiovascular disease, but also as a standard reference book for students and practicing physicians.

CARDIAC CLASSICS, A Collection of Classic Works on the Heart and Circulation with Comprehensive Biographic Accounts of the Authors. Fifty-two contributions by fifty-one authors. By *Frederick A. Willius, M. D., M. S. in Med., chief section of cardiology, The Mayo Clinic; professor of medicine, The Mayo Foundation for Medical Education and Research, The Graduate School, The University of Minnesota, and Thomas E. Keys, A. B., M. A., reference librarian, The Mayo Clinic; formerly Carnegie Fellow, The Graduate Library School, The University of Chicago.* Pp. 858, illustrated. The C. V. Mosby Co., St. Louis, Mo., publishers, 1941. Price \$10.

The authors of this collection of papers and books, which have been milestones in medical history, have done the difficult task of selection well. It must be remembered too that medical history is a valuable adjunct of clinical medicine and no less a one than Sir William Osler declared that he was a much better doctor after he became a student of medical history. One is certain to have a better understanding of the use of digitalis after reading Withering's "Account of the Foxglove." Incidentally, it is a remarkable tribute to Withering that our dosage and use today is so similar to that employed by him more than 150 years ago.

Among other famous names and subjects in the history of cardiology are William Stokes' "Observations on Some Cases of Permanently Slow Pulse"; Laennec's "Treatise on Mediate Auscultation"; Flint's

"Cardiac Murmurs," and Einthoven's original description of the electrocardiograph, to mention but a few.

The many fine illustrations add much to the book.

ELECTROCARDIOGRAPHY IN PRACTICE by *Ashton Graybiel, M. D., instructor in medicine, courses for graduates, Harvard Medical School; research associate, Fatigue Laboratory, Harvard University; assistant in medicine, Massachusetts General Hospital; and Paul D. White, M. D., lecturer in medicine, Harvard Medical School; physician, Massachusetts General Hospital, in charge of the cardiac clinics and laboratory.* Pp. 319 with 272 illustrations. W. B. Saunders Co., Philadelphia, publishers, 1941. Price, \$6.

The first part of the book consists of 142 electrocardiograms illustrating the normal electrocardiogram including 30 tracings showing normal variations, the remainder being devoted to the cardiac arrhythmias and tracings illustrating the characteristic changes in etiologic types of heart disease. The second part consists of 130 abnormal electrocardiograms with a short history, physical examination, and interpretation on the preceding page. As the authors state in the preface, these electrocardiograms "are presented in a heterogeneous fashion for practice in interpretation."

The method of presentation is similar to that used by the authors and their associates in their courses for graduates, including discussions of differential diagnosis, anatomy, physiology, and treatment. The clinical significance of the various electrocardiographic abnormalities is briefly discussed.

The principles and technic of electrocardiography, the description of the normal electrocardiogram and axis deviation are so clearly and concisely presented that the physician unfamiliar with electrocardiography will have little difficulty in comprehending these fundamental concepts.

In view of the tendency in recent years for many of those making electrocardiographic interpretations to ascribe clinical significance to certain electrocardiographic variations which are entirely unjustified, the inclusion of a complete series of normal variations, changes resultant from drug action, toxic states, and artefacts together with the normal findings in infancy and childhood are indeed welcome.

Except for the typical RST displacement in acute pericarditis most of the typical patterns are included.

This book is unreservedly recommended to every student of electrocardiography as a mine of information carefully selected and arranged by authors with great experience as clinicians, teachers, and investigators.

THE HEART IN PREGNANCY AND THE CHILDBEARING AGE by *Burton E. Hamilton, M. D., Cardiologist, since 1921, to The Boston Lying-In-Hospital, Boston, Mass., and K. Jefferson Thomson, M. D., associate physician, Metropolitan Life Insurance Company Sanatorium, Mount McGregor, N. Y.; research associate in medicine, Albany Medical College; formerly research fellow in obstetrics, Har-*

vard Medical School; formerly instructor in medicine, Cornell University Medical College. With a section entitled, "Delivery and Obstetrical After-Care of Cardiacs" by Frederick C. Irving, M. D., F. A. C. S., professor of obstetrics. Harvard Medical School, obstetrician in chief, Boston Lying-In-Hospital. Pp. 402. Little, Brown & Co., Boston, Mass., publishers, 1941. Price \$5.

The book is divided into three parts. The first part deals with the treatment of the cardiac case during the pre- and post-partum stages. The second part describes the physiology of circulation in the normal pregnancy and in pregnant women with heart disease. Part three gives a general discussion on the heart diseases in pregnancy and the childbearing age. The book is well illustrated with tables, charts, and photographs. The book should prove valuable to the general practitioner or to the obstetrician whose patient presents a problem of cardiac pathology.

THE AMERICAN COLLEGE OF PHYSICIANS, Its First Quarter Century, by *William Gerry Morgan, M. D., LL. D., Sc. D., M. A. O. P., Fellow (1916), councillor and regent (1916-29), governor (1929-32), secretary-general (1932-37), vice president (1937-38), and master (1940) of the American College of Physicians; professor of gastro-enterology and emeritus dean, Georgetown University School of Medicine, Washington, D. C.; former president American Medical Association. Published in Philadelphia, Pennsylvania, 1940. Price \$2.*

The American College of Physicians has celebrated its first quarter century of existence with an interesting memorial, a history of the College which has been published in book form, of which the Fellows may well be proud.

The story of the inception and development of the College has been well told. Dr. William Gerry Morgan, the historian, was well qualified for such a task, having been a Fellow since 1916 and having served in many capacities in the College.

A notable feature of the book is the beautiful cover, the fine nonglare paper, and the type which matches the finest printing.

QUICK REFERENCE BOOK FOR MEDICINE AND SURGERY, A Clinical, Diagnostic, and Therapeutic Digest of General Medicine, Surgery, and the Specialties, Culled Extensively and Intensively from Modern Literature, and Systematized. By *George B. Rehberger, A. B., M. D., Johns Hopkins University. Eleventh edition, revised. Pp. 1402, illustrated. J. B. Lippincott Co., publishers, 1940. Price, \$15.*

First copyrighted in 1920, this reference book of medicine has already passed through 10 editions, or about one every 2 years. Such an evidence of usefulness to the medical profession is enough to show its value. Many new developments have been added, including the use of nicotinic acid in pellegra, protomine insulin, paraldehyde in labor as an analgesic, and the increasing uses of sulfanilamide and its derivatives. There is a special section on anesthesia. Another most valuable feature is a dictionary of drugs covering action as well as dosage.

The illustrations include some most attractive full-page color plates. It is a good book on any doctor's bookshelf.

THE PRACTITIONERS LIBRARY OF MEDICINE AND SURGERY, 1940 Supplement. Supervising editor, *George Blumer, M. A. (Yale), M. D., David P. Smith, Clinical Professor of Medicine, Yale University School of Medicine; consulting physician to the New Haven Hospital.* D. Appleton-Century Co., New York, publishers. Price, \$8. Complete unit of 14 volumes, \$138.

An excellent book for the specialist or the general practitioner. Each contributor is a specialist in his field and the subjects presented cover the important new work in all branches of medicine and surgery. The book is well illustrated and easily read.

THE NEW INTERNATIONAL CLINICS, Vol. IV, December 1940 and Vol. I, March 1941, edited by *George Morris Piersol, M. D., professor of medicine, graduate school of medicine, University of Pennsylvania, Philadelphia, Penna.* Four volumes yearly, issued March, June, September, and December. Approximately 300 pages each. Illustrated. \$12 per year—annual subscriptions only. J. B. Lippincott Co., Philadelphia, Penna., publishers.

Besides the original contributions by well known authors in both volumes, the December volume contains 23 papers by members of the faculty of the University of Louisville School of Medicine; and the March volume has 13 articles by the faculty of the Yale School of Medicine. The articles are well written and present the current advances in medicine and surgery. The contributions, as could be expected from such contributors, are of the highest order and the articles should prove valuable to either the surgeon or internist.

SCABIES—CIVIL AND MILITARY by *Reuben Friedman, M. D., assistant professor of dermatology and syphilology, Temple University School of Medicine, Philadelphia, Penna.* Pp. 288. Froben Press, New York, publishers, 1941. Price, \$3.

This book is a treatise on scabies and gives a complete picture of the incidence and history of scabies. The various treatments are discussed in detail and the value of each is pointed out.

THE DIVISION OF PREVENTIVE MEDICINE

Captain C. S. Stephenson, Medical Corps, United States Navy, in charge

TOXIC EFFECTS OF ARSENICAL COMPOUNDS

AS ADMINISTERED IN THE UNITED STATES NAVY IN 1940 WITH SPECIAL
REFERENCE TO ARSENICAL DERMATITIS

By Captain C. S. Stephenson, Medical Corps, United States Navy, W. M. Chambers, Chief Pharmacist's Mate, United States Navy, and Mrs. Laura T. Anderson, Senior Clerk, Bureau of Medicine and Surgery

For the past 16 years medical officers of the Navy have been required to submit to the Bureau of Medicine and Surgery monthly reports of the number of doses of arsenicals administered and the reactions therefrom. This information has been compiled and published in the following United States NAVAL MEDICAL BULLETINS:

September 1925	April 1933	October 1936	October 1939
January 1927	October 1933	January 1937	January 1940
January 1929	October 1934	October 1937	October 1940
July 1930	January 1935	January 1938	January 1941
October 1931	October 1935	October 1938	
October 1932	January 1936	January 1939	

In table 1 is shown the number of doses of each arsenical administered in the year 1940, the reported reactions which occurred, and similar data for the 16-year period 1925-40. It is noted that in 1940 there was 1 reaction to 3,771 doses and 1 death to 124,445 doses. The fatal reaction occurred in a Samoan child who was receiving treatment for yaws. For the 16-year period 1925-40 there was 1 reaction to 1,539 doses and 1 death to 33,072 doses.

TABLE 1.—Arsenicals, U. S. Navy, 1940 and 1925-40—type of drug, reaction, and ratio of doses to reactions

	Number of doses administered	Reactions				Ratio of reactions to doses 1 to—	Ratio of deaths to doses 1 to—
		Mild	Severe	Fatal	Total		
Year 1940:							
Bismarsen	601	0	0	0	0	0	0
Mapharsen	81,411	10	4	0	14	5,815	0
Neosarsphenamine	36,976	8	10	1	19	1,946	36,976
Sulfarsphenamine	852	0	0	0	0	0	0
Tryparsamide	4,605	0	0	0	0	0	0
Total	124,445	18	14	1	33	3,771	124,445
16-year period 1925-40:							
Acetarson	971	1	0	0	1	971	0
Arsphenamine	41,558	27	14	1	42	989	41,558
Bismarsen	3,505	0	0	0	0	0	0
Mapharsen	203,100	24	12	0	36	5,642	0
Neosarsphenamine	1,338,889	626	311	50	987	1,357	26,778
Silver arsphenamine	586	0	1	0	1	586	0
Sulfarsphenamine	30,290	17	8	0	25	1,212	0
Tryparsamide	67,781	3	1	0	4	16,945	0
Total	1,686,680	698	347	51	1,096	1,539	33,072

¹ First administered during the year 1932.

² First administered during the year 1929.

³ First administered during the year 1935.

⁴ First administered during the year 1931.

TABLE 2.—*Proportion of reactions of various types, 1929-40*

Classification	Number of reactions	Percent of total reactions
Vasomotor phenomena.....	352	40.98
Arsenical dermatitis.....	326	37.95
Blood dyscrasias.....	44	5.12
Liver damage.....	38	4.42
Table reactions.....	26	3.03
Jarisch-Herxheimer.....	21	2.44
Reactions of minor importance.....	17	1.98
Gastrointestinal.....	17	1.98
Hemorrhagic encephalitis.....	8	.93
Optic neuritis.....	3	.35
Arsenical neuritis.....	2	.23
Acute renal damage.....	2	.23
Border line, hemorrhagic encephalitis.....	1	.12
Liver damage (doubtful reaction).....	1	.12
Vascular damage (probable adrenal hemorrhage).....	1	.12
Total.....	859	100.00

ARSENICAL DERMATITIS

Dermatitis in some form was observed in 18, or 55 percent of the total reactions in 1940, as compared with 44 percent for 1939. The type of lesion was erythematous in two instances, exfoliative in eight, macular in three, maculopapular in four, and urticarial in one. The reactions were classified as nine mild, and nine severe.

MILD REACTIONS

The nine mild reactions occurred after the following number of injections: One after the first injection, three after the third, one each after the fourth, ninth, twelfth, fourteenth, and twenty-ninth.

The interval between injection and appearance of symptoms varied from 1 hour to 4 days. The length of time required for complete recovery varied from 2 days to 17 days.

NEOARSPHENAMINE

Case 1—1940.—Eight days after exposure to infection on June 8, 1940, this patient developed an initial lesion on frenum of penis. Upon examination on June 25 a pale pink macular rash was noted on the chest and abdomen. A darkfield examination of the lesion was positive for *Treponema pallidum*. A Kahn blood test was 4 plus.

Arsenical treatment began with a 0.3 gram injection of neoarsphenamine on June 29, followed by a 0.45 gram injection on July 3, and a 0.5 gram injection on July 7. Four injections of bismuth subsalicylate were given as concurrent treatment.

Five hours after the last injection of neoarsphenamine on July 7 the patient developed a generalized maculopapular rash, nausea, and vomiting. Temperature, 103.8° F. Two 1-gram injections of sodium thiosulphate were administered intravenously. The rash faded rapidly, and the temperature gradually subsided under treatment. Recovery in 15 days.

Case 2—1940.—One month after exposure to infection on November 10, 1939, this patient was given a diagnosis of syphilis because of a 4 plus Kahn blood test.

From December 12, 1939, to January 30, 1940, he received eight injections of neoarsphenamine, a total of 3.75 grams, and from January 9 to April 2, 1940, 13 intramuscular injections of bismuth subsalicylate, a total of 1.69 grams, as concurrent treatment.

The second course of arsenical treatment began with a 0.3 gram injection of neoarsphenamine on April 9, followed by 0.45 gram injection on April 17 and 0.5 gram injections on April 23 and April 30.

Forty-eight hours after the last injection the patient developed a generalized erythematous, papular itching of the skin. Laboratory findings: RBC, 4,290,000; WBC, 7,450; hgb., 90 percent; bands, 8; segs, 53; lymphs, 28; eosins, 5; monos, 6.

Two 1-gram injections of sodium thiosulphate were administered intravenously on May 3 and May 4. Recovery in 11 days.

Case 3—1940.—After exposure to infection on August 1, 1932, this patient developed an ulcer on the penis. A darkfield examination was positive for *Treponema pallidum*. Kahn blood tests were 4 plus on August 24 and October 27, 1932.

From August 23, 1932, to August 22, 1934, he received 28 injections of neoarsphenamine (total amount in grams not reported). Concurrent treatment consisted of bismuth injections, mercury inunctions, and 2 courses of potassium iodide.

A Kahn blood test was 4-plus on July 24, 1940. Arsenical treatment was again instituted on July 25 with a 0.15 gram injection of neoarsphenamine. Twelve hours after the injection the patient developed a temperature of 102° F. and a macular rash on the face, arms, trunk, and upper thighs. WBC, 6,650; RBC, 4,850,000; hgb, 90 percent; bands, 2; segs, 68; lymphs, 25; monos, 3; eosins, 2.

Five grains of sodium thiosulphate were given on July 26, 10 grains on July 27, and 15 grains on July 28. Recovery in 4 days.

MAPHARSEN

Case 4—1940.—This patient developed an initial lesion on abdomen and suprapubic region following exposure to infection on August 4, 1940. A secondary rash developed over arms and chest. Darkfield examination was not obtained. A Kahn blood test was 4 plus.

Arsenical treatment began with a 0.03 gram injection of mapharsen on September 10, followed by 0.06 gram injections on September 17 and 24.

Two and one-half hours after the last injection of mapharsen the patient complained of chills, fever, and vomiting. Temperature, 103° F.; pulse, 120; respiration, 20; WBC, 7,400; RBC, 4,210,000; hgb, 90 percent; bands, 1; segs, 74; lymphs, 24; mono, 1.

About 5 hours after the last injection a generalized maculopapular rash appeared. The rash was most prominent on chest and abdomen.

Symptoms subsided rapidly with no specific treatment administered. Recovery in 2 days.

Case 5—1940.—Following exposure to infection on April 30, 1940, this patient developed a primary lesion on coronal sulcus of the penis. On May 23 a darkfield examination of the lesion was positive for *Treponema pallidum* and a Kahn blood test was 4 plus.

Arsenical treatment began with a 0.04 gram injection of mapharsen on this date, followed by 0.06 gram injections on May 30 and June 8.

Eighty hours after the last injection the patient developed a generalized itching and diffuse punctate erythema of trunk and arms.

He was given force fluids, systemic alkalization, and a high carbohydrate diet. Recovery in 4 days from onset of symptoms.

Case 6—1940.—On January 8, 1940, 1 month after exposure to infection, this patient developed a primary lesion at the junction of mucous membrane and skin just lateral to the urethra. A darkfield examination on this date revealed a few spiral organisms characteristic of *Treponema pallidum*. A Kahn blood test on January 10 was 4 plus.

Arsenical treatment began with 0.02-gram injections of mapharsen on January 10 and 13, or 0.04 gram injection on January 16, and a 0.06 gram injection on January 23, a total of 0.14 gram.

Four-days after the last injection the patient developed a maculopapular rash on the face, arms, and thorax, confluent in patches with slight excoriation in places. Two 1-gram injections of sodium thiosulphate were given intravenously. Recovery in 8 days.

Case 7—1940.—After exposure to infection on April 24, 1940, this patient developed a primary lesion and bilateral inguinal and posterior cervical adenopathy. A darkfield examination was positive for *Treponema pallidum* on May 11, 1940.

He was given 8 injections of mapharsen, a total of 0.39 gram, and 4 injections of bismuth subsalicylate in oil, a total of 0.52 gram.

A second course of arsenical treatment began with a 0.03 gram injection of mapharsen on October 8, 1940. Nine hours after the injection the patient developed headache, dizziness, and a maculopapular eruption.

Kahn blood tests on October 8 and 24, 1940 were 4 plus. CBC and RM was within normal limits.

One gram of sodium thiosulphate was given daily for 3 days. Recovery in 17 days.

Case 8—1940.—Three weeks after exposure to infection on September 17, 1939, this patient developed a primary lesion on prepuce of penis. A Kahn blood test was 4 plus. From October 6 to November 22 he received 8 injections of neoarsphenamine, a total of 3.35 grams, and from October 30, 1939, to January 25, 1940, 0.96 grams of bismuth subsalicylate. A severe exfoliative dermatitis followed the last injection of neoarsphenamine. (See Case 13—1939, Naval Medical Bulletin, October 1940).

The second course of arsenical treatment began with a 0.03 gram injection of mapharsen on June 26, 1940 and 24 hours later the patient noticed an intense itching of the skin over the entire body. Examination revealed lacrimation of the eyes, and marked injection of the mucosa of the nose and throat, anterior and posterior cervical lymphadenopathy. There was a generalized macular eruption which became confluent around the face and in the folds of the body. Urinalysis showed a faint trace of albumin and was positive for occult blood.

One gram of sodium thiosulphate was given intravenously. The skin condition developed into an exfoliative dermatitis. Recovery in 4 days.

Case 9—1940.—This patient was exposed to infection in April 1939, and developed a penile lesion and adenopathy. Darkfield examinations and Kahn blood tests were negative. On June 8 fluid obtained from a gland puncture was positive for *Treponema pallidum*.

From June 6 to July 11, 1939, the patient received 6 injections of neoarsphenamine, a total of 3.30 grams; from July 18 to October 4, 4 injections of mapharsen, a total of 0.24 gram; and from July 25 to December 27, 16 injections of bismuth subsalicylate, a total of 2.08 grams. A mild skin reaction followed the last injection of neoarsphenamine.

The second course of arsenical treatment consisted of 0.06 gram injections of mapharsen on December 27, 1939, January 2, 9, 16, and 23, 1940. A mild urticarial rash developed 1 hour after the injection administered on January 16 and disappeared the next day. WBC, 12,300; polys, 61; lymphs, 37; bands, 1; immature, 1. Because of the appearance of the rash it was believed this might be due to food eaten. One-gram injections of sodium thiosulphate were administered on January 16 and 17.

One hour after the administration of 0.06 gram of mapharsen on January 23 an eruption of itching wheals developed over the trunk and arms. The eruption was red in places and especially thick in axillae, over face and palms, and antecubital spaces. WBC, 12,750; segs, 66; lymphs, 22; bands, 5; eosins, 4; immatures, 3. One-gram injections of sodium thiosulphate were administered on January 23 and 26. Recovery in 3 days.

SEVERE REACTIONS

The nine severe reactions occurred after the following number of injections: Two after the eleventh; and one each after the second, eighth, ninth, tenth, fourteenth, twenty-fifth, and one hundred eighteenth.

The interval between the injection and appearance of symptoms varied from 3 hours to 5 days. The length of time required for recovery varied from 19 to 178 days.

NEOARSPHENAMINE

Case 10—1940.—After exposure to infection on June 1, 1940, this patient developed a sore on the skin of the penis at right lateral aspect overlying the coronal sulcus. Darkfield examinations were negative.

Arsenical treatment began with a 0.3 gram injection of neoarsphenamine on July 22, followed by a 0.45 gram injection on July 29. Two injections of bismuth were given as concurrent treatment.

Two days after the last injection of neoarsphenamine the patient developed enlarged postcervical glands and generalized adenopathy. Temperature 104.8° F. The entire body, extremities, and face were covered with a bright red, macular, nonitching rash which in appearance was different from a luetic eruption.

August 1: The skin lesions are confluent and cover the entire body, being almost hemorrhagic in spots. Temperature 105° F. The glands in the back of the neck were painful and the patient complained of headache and some abdominal discomfort. Sodium thiosulphate was given intravenously and by mouth.

August 5: Progress entirely satisfactory. Temperature has been normal for 48 hours and there are no subjective complaints.

August 7: Rash fading. Characteristic desquamating lesions of arsphenamine poisoning over shins. Conservative treatment with emollients and desquamating agents begun. Symptomatic treatment carried out with daily injections of sodium thiosulphate. Recovery in 28 days.

Case 11—1940.—After exposure to infection on September 18, 1940, this patient developed multiple lesions on coronal sulcus of the penis. Darkfield examinations of the lesions were negative for *Treponema pallidum*. A Kahn blood test on November 6 was 4 plus.

Arsenical treatment began with a 0.45-gram injection of neoarsphenamine on November 6, followed by 0.6-gram injections on November 8, 13, 20, 27, December 4, 11, and 18. Five days after the last injection the patient was admitted with generalized erythematous areas with desquamation, over the face, arms, hands, and legs. Red-blood count, 4,500,000; white-blood count, 9,000; bands, 3; segs, 52; lymphs, 36; eosins, 4; monos, 5. He was given 1 gram of sodium thiosulphate

daily for 16 days, and colloid baths, olive-oil inunctions, and low carbohydrate diet. The patient was returned to duty status under treatment, 20 days after onset of symptoms.

Case 12—1940.—After exposure to infection on October 12, 1939, this patient developed a pinkish maculopapular rash on the neck, trunk, arms, and thighs, and macules on soles of the feet. The inguinal glands were enlarged, discrete, and firm. There were venereal warts on the frenum, surrounded by an area of induration. A darkfield examination of this area was negative. Kahn blood test and Ide test were 4 plus.

Arsenical treatment began on December 27, 1939, with a 0.3-gram injection of neoarsphenamine, followed by nine weekly 0.5-gram injections, a total of 4.8 grams. He received 6 injections of a bismuth compound as concurrent treatment.

Two days after the last injection of neoarsphenamine (February 21, 1940), the patient's ankles were swollen and he stated that this condition had existed for 6 days. Physical examination failed to reveal any cause for this edema, and since there was a suggestion of thickening and coarsening of the skin it was believed that this might have been the beginning of an arsenical reaction. This was further supported by an eosinophilia of 18 percent. On March 3, a more marked coarsening of the skin around the neck and on the hands and forearms was noted. Two 0.5-gram injections of sodium thiosulphate were given intravenously. The skin conditions developed into an exfoliative dermatitis which showed steady improvement under treatment. Recovery in 22 days.

Case 13—1940.—After exposure to infection on June 5, 1940, this patient developed a primary lesion which was positive for *Treponema pallidum*. A Kahn blood test was 4 plus.

From July 23 to September 30 he received 5.7 grams of neoarsphenamine, and from August 15 to September 19, 0.78 gram of bismuth salicylate as concurrent treatment.

The second course of arsenical treatment began with 0.6-gram injections of neoarsphenamine on September 17 and 22, 1940.

Three hours after the last injection of neoarsphenamine, symptoms of generalized itching of the skin with urticaria appeared. WBC, 14,500; RBC, 4,500,000; Hgb, 8 percent; segs, 66; bands, 1; monos, 4; lymphs, 21, eosins, 8.

A severe exfoliative dermatitis developed, associated with itching and urticaria involving the face, trunk, arms, and lower extremities. The lips were cracked and swollen. The skin over the hands and fingers and extending upward over the flexor surfaces of the arms was cracked, inflamed, and exuded serum.

The patient was given 1 gram of sodium thiosulphate intravenously twice weekly and 1 gram by mouth daily. Boric-acid ointment, with 1 percent phenol, was applied to the skin, followed by bran baths and the application of olive oil. The urticaria was controlled by 0.5 cc. intramuscular injections of adrenalin.

The skin condition gradually improved under treatment. Recovery in 113 days.

Case 14—1940.—After exposure to infection on October 1, 1939, this patient developed a lesion on shaft of the penis which was positive for *Treponema pallidum*. A Kahn blood test was 4 plus.

From October 14 to December 6, 1939, he received 9 injections of mapharsen, a total of 0.54 gram; from January 13 to March 8, 1940, 8 injections of neoarsphenamine, a total of 4.05 grams; and from December 6, 1939, to January 8, 1940, 7 injections of a bismuth compound as concurrent treatment. From January 13 to March 8, the patient was given a third course of arsenical treatment, consisting of 8 injections of neoarsphenamine, a total of 4.05 grams. During the administration of the fifth injection of the third course (Feb. 13, 1940) there was a little leakage around the vein. The skin about the area turned red and

he had some pain in the vicinity. This cleared up in 1½ weeks. On the day following the last injection (0.6 gram—Mar. 8, 1940) the patient noticed a rash on the arms and legs. Examination revealed weakness, anorexia, slight edema of the face, pruritis, and an erythematous exfoliative dermatitis involving the extensor surfaces of the upper extremities, face, and neck. Several areas were infected as a result of scratching, and these discharged semi-purulent material. No evidence of renal complications was present. Temperature, 99° F.; pulse, 82; respiration, 20. Temperature increased and reached a maximum of 101° F., becoming septic with morning remissions.

Treatment for this condition consisted of daily colloidal baths and starch powder. The lesions on the chest and face cleared rapidly under treatment but the hands and feet remained very sore, deeply cracked, and painful.

Iron therapy in the form of Blaud's pills was started on April 1 with daily gelatin baths followed by unguentum aqua rosa. There was a mixture of fungus infection present as well as exfoliative dermatitis. All of the lesions healed except the lower legs on which a severe erythematous scaling dermatitis remained. Denudation, however, was not present and edema not apparent. The legs were painted with 1 percent gentian violet. Treatment began with oatmeal baths and boric acid ointment. Marked improvement was evident, and the patient recovered in 157 days from onset of symptoms.

Case 15—1940.—This patient was given a diagnosis of syphilis because of a 3-plus reversible Kahn blood test on December 9, 1939, a 4-plus reversible Kahn blood test on December 17, and a 4-plus Kahn blood test on December 28. Diagnosis was corroborated by a history of chancroidal infections of penis and lymph nodes in 1931 and 1932, scars in both groins and on penis, bilateral inguinal and postcervical lymph nodes, and palpable epitroclear lymph nodes. The reversible Kahn blood test indicated an old infection. A seropositive diagnosis of latent syphilis, probably of 8 or more years' duration, was established on January 9, 1940.

A preliminary course of bismuth subsalicylate in oil was begun on January 9, 1940. Thirteen 0.2 gram intramuscular injections at weekly intervals were administered between this date and May 7. Arsenical treatment began with a 0.3 gram injection of neoarsphenamine on March 12, 1940, followed by a 0.45 gram injection on March 19, and weekly 0.6 gram injections for 6 weeks, a total of 4.95 grams.

The patient was admitted to the sick list on the day of the ninth injection of neoarsphenamine complaining of a generalized itchy rash of about a week's duration. The rash was urticarial, erythematous (resembling sunburn in places), and measly and miliarial in type.

The patient had been standing 4-hour daily watches on a signal tower and had been exposed to the sun rays for 44 hours. He first noted prickly heat and excessive perspiration over the entire body, sunburn of the face and hands, and a "blotchy" red eruption with itching.

Physical examination revealed a stippled erythematous rash, especially of the flexor surfaces of the elbows and knees, also patches of eczematous-type dermatitis varying in size from one-half to two inches in diameter. The skin was a dusky color, dry, indurated, hard, and leathery.

May 10: RBC, 4,330,000; WBC, 5,350; hgb, 95 percent; bands, 5, segs, 65; lymphs, 21; monos, 5; eosins, 3; basos, 1. Except for a rise in temperature to 100° F. one evening he has had no fever. There is beginning exfoliation and desquamation. No jaundice or diarrhea. Urinary output is satisfactory.

Four intravenous injections of sodium thiosulphate were administered between May 10 and May 13, a total of 3.25 grams.

May 14: Temperature was 99.4° F. the previous night. Fluid intake 2,170 cc.;

output, 1,640 cc. Appetite poor. RBC, 4,520,000; WBC, 6,700; hgb, 90 per cent; differential, normal except for 5 percent eosinophiles.

May 18: Urinalysis: specific gravity, 1.022; sugar and albumin, negative; 2 WBC per HDF; few epithelial cells. The skin is desquamating freely, especially over the back and flexor surfaces; the hands and soles of the feet are hard and brown; and the face is somewhat puffy.

June 1: The patient transferred to hospital for further treatment.

June 4: After consultation it was deemed advisable to continue antiluetic therapy with 0.1 gm. of bismuth biweekly, and 30 grs. of potassium iodide t. i. d. in ascending doses until untoward symptoms develop.

June 8: The patient has been on oatmeal colloidal baths t. i. d., followed by boric ointment. Condition of skin improving. Edema of ankles persists.

June 14: Edema of the ankles prominent. This is most prominent in the evening and tends to disappear during the night. Serum proteins are normal. At present it is questionable whether this edema is cardiac in origin or is an inflammatory type due to the marked dermatitis present in this area. Skin clearing rapidly in all parts of the body. General condition good.

July 31: All medication discontinued. The lesions are dry and scaly.

August 7: Rash is acute and oozing and is localized to the legs. Diagnosis of poisoning, therapeutic, acute, neoarsphenamine, syphilis, changed this date to eczema.

August 30: Much improvement. Only remaining defect is a thinness of skin over site of lesion which is characterized by redness when the legs are elevated and cyanosis when the legs are dependent. Ace bandages used to aid venous return.

September 12: Skin and legs have cleared considerably, however, marked residual pigmentation remains. Massage with ointments.

September 21: Consultation with syphilologist to ascertain advisability of discontinuing potassium iodide. Occasional papulo-erythematous lesions on legs around the site of previous dermatitis, probably due to iodides. Potassium iodide discontinued.

October 5: Sores on both legs are very indolent. Given viosterol, heat lamp, and gentian violet to ulcers.

October 14: Multiple ulcers on both lower extremities and 3 small carbuncles on posterior surface of neck. This condition is not related to syphilis. Patient placed on sulfathiazole by mouth, and hot compresses applied.

All lesions gradually healed, and the patient was returned to duty 178 days after admission with the recommendation that alternate courses of bismuth and mercury rubs be given.

MAPHARSEN

Case 16—1940.—This patient was exposed to infection on April 7, 1937, and developed an initial lesion behind coronal sulcus of the penis on April 13. A darkfield examination of the lesion was positive for *Treponema pallidum*. A Kahn blood test was positive.

From April 22, 1937 to April 16, 1940, he received 62 injections of mapharsen, a total of 3.415 grams; 46 injections of neoarsphenamine, a total of 22.56 grams; 58 injections of a bismuth compound, a total of 102 cc.; and 20 injections of mercury, a total of 20 cc.

Kahn blood tests during the 3-year period of treatment were positive from time to time. The Kahn test on February 27, 1940, was 4-plus.

The last course of arsenical treatment began with a 0.04 gram injection of mapharsen on May 2, 1940, followed by 9 weekly injections of 0.06 gram each, a total of 0.58 gram. The last injection was given on July 5; 4 days later

examination revealed a maculopapular and pustular rash over the toes and in spots on the fingers with blister formation and much peeling and scaling. There were large macular blotches beginning to extend up the legs. The only complaint pertaining to rash was a sensation of "burning."

About 2 weeks before admission the patient was being treated for trichophytosis of the feet, which was apparently getting worse. Treatment for this condition continued and no thought was given to possible arsenical poisoning.

July 27: It is apparent that this condition is spreading rapidly and the general appearance is that of beginning exfoliative dermatitis. Sodium thiosulphate given intravenously. Sodium bicarbonate baths and vaseline to hands and feet. The dorsal surfaces of the hands are covered with large blebs and the eruption is spreading up the thighs and arms with a small amount on the trunk.

July 28: Blebs have broken and eruption seems to have taken on a more dry character. Eruption is now more or less confluent over arms, legs, and trunk. Sodium thiosulphate administered and sodium bicarbonate baths continued. Vaseline applied for dryness of the eruption.

July 29: Sodium thiosulphate given intravenously. It is the opinion that this patient's condition represents an acute arsenical dermatitis due to the therapeutic administration of mapharsen. The skin lesions gradually subsided under treatment. Recovery in 20 days from onset of symptoms.

Case 17—1940.—This patient has had 3 syphilitic infections, the third being incurred on July 17, 1939, while the patient was under active antiluetic treatment. The current infection was diagnosed by a positive darkfield examination, and a positive 4-plus Kahn blood test.

From July 20 to September 17, 1939, the patient received 1.95 grams of neoarsphenamine. He had no difficulty in withstanding routine treatment consisting of alternate courses of neoarsphenamine and bismuth salts up to the last week in July 1939 when he developed an immediate (Herxheimer type) reaction following a routine neoarsphenamine injection. The following week a more severe reaction developed, consisting of urticarial wheals, flushing of face and tingling over body. Red and white blood counts remained normal during the following week. Treatment was changed to mapharsen as there was no indication of arsenical poisoning. The reaction was attributed to the chemical structure of the neoarsphenamine, i. e., the patient appears to have developed an idiosyncrasy to benzene ring compounds. He received 3 injections of mapharsen, a total of 0.112 gram, without reaction or evidence of arsenical poisoning, the last injection being given in September 1939. The patient was granted leave and was advised to report to a naval station for further treatment. He had no treatment for 3 weeks, and when seen on January 8, 1940, it was thought that he had secondary syphilis. There were lesions on the trunk and abdomen, accompanied by mild itching and mucous patches in the throat. He was given a 0.04-gram injection of mapharsen on January 9, an injection of bismuth on January 14, and a 0.06-gram injection of mapharsen on January 14. After this injection the rash began to fade and the itching subsided. Several days later he developed a generalized dermatitis accompanied by intense itching. Examination revealed a macular rash with slight elevation above level of surrounding skin, involving areas to wrist, trunk, legs, and shaft of penis. The eruption was pronounced and severe about the flexor surfaces, axillary, and groin. There was a tendency toward dryness and silvery sheen about some lesions. Treatment for this condition consisted of 19 daily 1-gram injections of sodium thiosulphate, 20 units of liver extract, and sponge bath followed by cold cream applied locally. The skin condition gradually improved under treatment and recovery was considered complete in 19 days.

Case 18—1940.—After exposure to infection in December 1939, this patient developed a slight abrasion on the coronal sulcus. Five consecutive darkfield examinations were negative, and the lesion healed in 5 days. On March 11 and 18, 1940, Kahn blood tests were 4-plus.

From March 20 to May 29, 1940, he received 12 injections of neoarsphenamine, a total of 6.75 grams, and 10 injections of bismuth subsalicylate as concurrent treatment.

The second course of arsenical treatment began with 0.03 gram of mapharsen on September 5, followed by a 0.06-gram injection on September 12. Five days later an examination revealed a diffuse moderate exfoliative dermatitis over arms, chest, and back. The patient stated that the itching had been first noted on the day after the second injection.

Sodium thiosulphate was given intravenously, and calamine was applied locally. The skin condition gradually improved under treatment. Recovery in 36 days.

SUMMARY

In 1940 medical officers of the Navy administered 124,445 doses of arsenicals and reported the occurrence of 33 reactions therefrom. Of these reactions 18 were arsenical dermatitis; a ratio of one case of dermatitis to 6,914 doses. Of interest in connection with the etiology of arsenical dermatitis is the number of instances in which premonitory signs were noted. These signs are repeated below and serve to indicate the necessity for careful examination and questioning of each patient before administering an arsenical.

Case 9.—A mild urticarial rash developed after the fourth injection of mapharsen. An eruption of itching wheals followed the fifth injection given 1 week later.

Case 12.—Swelling of the ankles and thickening and coarsening of the skin followed the ninth injection of neoarsphenamine. Eleven days later an exfoliative dermatitis developed.

Case 17.—During a course of neoarsphenamine a Herxheimer reaction occurred. A substitution of mapharsen was made and eight injections were given without ill effects. Three months later a generalized macular rash followed an injection of mapharsen.

Case 18.—A mild rash developed after the first injection of mapharsen. An exfoliative dermatitis followed the second injection given 1 week later.

OBSERVATIONS ON MOSQUITO AND MALARIA CONTROL IN THE CARIBBEAN AREA ¹

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In October 1940, a force of United States Marines was sent to one of the islands of the West Indies. The mosquito population offered

¹ Because of military necessity, the exact location where this work has been done has not been given. At a later date, anyone interested might obtain additional information from the Editor.

one of the most urgent medical problems, not only from the standpoint of personal annoyance, but also from the standpoint of health, since the vectors of all the mosquito-borne diseases were present. At sundown the mosquitoes came in such swarms that head nets and gloves had to be worn by personnel on guard duty. All but necessary work had to be suspended and unengaged personnel took to the cover of mosquito nets on their bunks. Even during daylight hours mosquitoes were present but in considerably less numbers. Identification of these adult mosquitoes showed a great majority to be of the nuisance type (*Psorophora columbiae* and *Aedes taeniorhynchus*) although a few anophelines, primarily *albimanus*, were present.

As a result of studies begun in 1936, Carr, Melendez and Ros (1) had shown malaria to be endemic in the entire province, with epidemics not uncommon when local conditions favored the breeding of anophelines capable of transmitting the disease. However, until the winter of 1940-41 malaria had never been a serious problem since the station was small and fairly well removed from the neighboring towns. Relatively few native workmen were employed and all personnel were housed in well-screened quarters. Lack of entertainment facilities ashore other than swimming, hiking, and baseball, created no "liberty" problem for ships of the fleet when they arrived, as most men returned aboard before nightfall.

The decision to develop this station into a fairly large and well-equipped operating base, completely changed this picture. In the summer of 1940 construction work was started by a civilian contractor who brought in better than 3,000 native workmen from surrounding towns. Shortly thereafter it was decided to use a portion of the reservation as a training base for the marines. In less than 8 months, the station's population increased more than 15 times its size.

The marine camp was built on several small peninsulas extending into the bay and located approximately 2½ miles in a direct line, and 5 miles by road, from the naval station. The troops were quartered in unscreened tents with each man being furnished a mosquito net for his bed. Of the native workmen employed on the station, all but approximately 1,200 commuted each day between neighboring towns. Those quartered on the station lived, for the most part, in "barracones" (open thatched roof shelters) where little or no attempt was made to protect the individual from mosquitoes.

In addition to the marked increase in the local population, with the possible induction of malarial carriers among the native workmen, new mosquito breeding areas were created through activities associated with the construction program. Excavation, leveling off

and filling, and road building led to the formation of many pools during the unseasonable increase in rainfall. (See table I.)

In December several cases of malaria developed in the marine personnel. Although oiling as a mosquito control measure in the neighborhood of the camp had been practiced from the day the marines arrived, it became apparent with the development of these cases that more active measures directed toward the control of this disease would have to be taken.

The control of malaria in an endemic area is no small problem; its eradication almost impossible. Clark (2) makes the statement that "mass treatment or the treatment of carriers selected by microscopic surveys will not eradicate malaria from any tropical region that we have known, regardless of what drugs are used." However, we felt that an attempt to control the anopheline mosquitoes combined with the identification and treatment of the carriers, was necessary to reduce the malarial incidence rate. It is reasonable to suppose that if the number of vectors is reduced and the peripheral blood of carriers sterilized by medication, the chances of infection are reduced directly according to the success of these efforts.

For the control of mosquitoes, oiling, drainage, and clearing were the methods most applicable to this area and were those used. For the detection of carriers, a thick-film survey of all native workmen living on the station was undertaken according to the method outlined by Barber and Komp (3).

From the increasing number of reports of accidental transmission of malaria by blood donors who have had malaria previously, but whose blood at the time was free of parasites, we realized the inadequacy of the thick-film study in the detection of all carriers. Clark (4) has very aptly called our attention to the fact that "a few drops of blood do not tell us all about the volume of blood in an individual insofar as the presence or absence of a few malarial parasites is concerned." But this method is the best that has yet been devised for community work and is an excellent time-saving procedure.

Further, the possibility that recent recruits from malarial sections of the United States, as well as men with longer service and considerable tropical duty, might also be carriers was fully recognized. However, the fact that these men are under close medical supervision and take no medication, except on the order of a medical officer, made us feel that as a control measure, thick-film studies on the marines, while desirable, was not a problem of first importance. This was later proved at the end of the third native survey by a survey of the First Marine Aircraft Group, an organization of older

men who have had considerable tropical duty. In this survey the blood of 701 marines was examined and only two positive cases were found; a carrier rate of 0.29 percent as compared to a carrier rate of 2.49 percent which was found among the natives.

The shore line of the bay near the camp is broken by marshes and coves, and is fringed with a heavy growth of mangroves. The marshes and coves are separated by irregular claw-like peninsulas which jut into the bay about 20 to 30 feet above the high-tide level. The marine camp was located on several of these peninsulas on the south side of the bay. The Naval Station is built on similar terrain and extends somewhat further inland, but few places on it have an elevation of more than 50 to 100 feet. This feature of the terrain is of fundamental importance in the Caribbean area since a direct relationship between altitude and areas of endemic malaria has been established; those areas along the low-lying coast or below 1,000 feet elevation having a high incidence of the disease (5) (6).

Extending inland from the coves are flat valleys that drain relatively short watersheds. The valleys terminate in sand flats or broad stretches of low ground covered with thick vegetation. After heavy rains the ground becomes marshy and provides excellent breeding areas for mosquitoes. The streams usually become dry between rains. Absorption does not appear to play a great part in the disposal of storm water, due to the impervious nature of most of the subsoil. Also, in the low flats, back seepage of the bay water further hinders drainage.

Rain water collected at elevations less than 10 feet above sea level have been observed to become strongly brackish, and at higher levels there is still a trace of salt. In some of the valleys the slope is so gradual that the 10-foot contour is nearly a mile inland from the high water mark. A great deal of this terrain must have been below sea level at one time to effect this wide distribution of salt in the soil.

There is no well-established rainy season as exists in some tropical regions. The annual rainfall as well as the distribution of the rains is subject to considerable variation. The accompanying table (table I) shows the average monthly precipitation for the past 8 years, compared with the rainfall during the months that the marine division was there. The greater part of each month's rain was concentrated into periods of 2 to 6 days. From the above it would be expected that the mosquito population would show a variation correlated with the periods of heavy rains, and that the predominating species would be brackish water breeders. Both of these expectations were realized.

TABLE I.—*Weather data*

Month	8-year average			Rainfall (inches) October 1940- March 1941
	Temperature		Rainfall (inches)	
	Maximum	Minimum		
January.....	87.0	62.7	0.23	2.04
February.....	86.5	61.5	.65	7.61
March.....	88.0	62.9	.80	3.77
April.....	88.7	65.6	.10	
May.....	89.4	68.8	2.48	
June.....	90.4	61.4	4.50	
July.....	92.4	71.4	1.35	
August.....	92.4	71.4	2.87	
September.....	91.9	71.4	2.00	
October.....	91.0	69.5	9.80	11.73
November.....	89.4	66.9	2.96	1.90
December.....	88.3	63.5	.86	3.71

MEASURES FOR THE CONTROL OF MOSQUITOES

The medical department personnel from the First Marine Division engaged in mosquito control work consisted of one medical officer and from two to six hospital corpsmen, to whom were added marines as needed. The usual grouping were two crews of one hospital corpsman and three marines each. Each crew was provided with a light truck, knapsack sprayers, larvae collection equipment, rubber boots, and shovels. All men were taught to distinguish anopheline larvae, and they were instructed to bring in specimens whenever found.

The medical officer's field equipment included a topographical map of the area, pencils and notebook, and material for taking and preserving larvae and adult mosquitoes. The latter material was largely improvised. A satisfactory dipper was made by securing a tin can to a pole. A larvae dip-net was made from mosquito netting on a heavy wire frame with a one-ounce bottle secured at the apex of the net. This net concentrates the larvae from approximately a gallon of water into the one-ounce bottle and thus facilitates the search in bodies of water where larvae are scarce.

An improvised butterfly net was made for adult mosquitoes. This net is of limited use, being adapted to sweeping through marsh grass to catch the mosquitoes lurking there. A collecting tube is needed for capturing mosquitoes while they are resting on tents, screens, or persons. Such a tube was made by filing the bottom from a Kahn test tube and fitting onto it an 18-inch length of rubber tubing with a single layer of coarse mesh gauze at the junction. With the rubber tube in the mouth, it was simple to suck a mosquito up into the glass tube and blow it out into a killing bottle. The killing bottle was a 4-ounce widemouth screw top bottle in the bottom of which was a layer of chopped rubber bands soaked with chloroform and covered with a layer of cotton and filter paper.

Ten percent silver nitrate was carried in a dropping bottle for a rough salinity test. When dropped into sea water this concentration

of silver nitrate instantly forms a solid curd of silver chloride, hardly larger than the drop. In water having a lower concentration of salt there is increasing dispersion of the silver nitrate before the cloud of precipitate is formed. Match boxes served as containers for adult mosquitoes while any type of stoppered bottle was satisfactory for larvae. We used the larvae preservative recommended by Dunham (7) consisting of a 10 percent formalin solution containing 2 percent of glycerin and 0.5 percent of borax. A hand lens is a most desirable piece of equipment which we did not have; a 2× reading glass helped in a degree to fill the need.

The object of the mosquito survey was to determine (1) what malaria transmitters were present; (2) the location of the breeding places of the transmitters; (3) the breeding places of other than malaria-transmitting mosquitoes, and (4) the most feasible methods for mosquito control. Initially it appeared more practicable to identify the anophelines in the larval stage so the survey began with a study of water collections. Accordingly, the following types of water collections were investigated: running streams, storm water pools, marshes, crab holes, mangrove swamps, man-made holes around construction projects, artificial containers such as water tanks, old tin cans, and bottles and bromeliad plants. The area we attempted to cover was within 1 mile of the camp sleeping quarters, as this is the usually given extent of anopheline flight.

Constantly running streams are few in this vicinity but there are great numbers that run for varying length of time after a period of rains. A detailed reconnaissance was made with the object of locating all dry stream beds as well as existing pools. In following up dry stream beds, pot holes and low areas that might become marshy, were noted. This information was mapped and recorded in notes and later proved its worth in the time saved in locating pools to be oiled after rains. The number of pools remaining after rains may be overwhelming and unless oiling efforts are systematized, they are bound to be ineffectual. Even so, we were unable to oil all pools in time to prevent the emergence of some of the first crop of mosquitoes which occurred 7 or 8 days after the beginning of a rain.

One typical small stream, harboring *Anopheles albimanus* and *A. crucians*, runs for about 200 yards in the upper levels of its bed and except after a rain is dry for the last half mile before reaching the bay. It is exposed to the sunlight and varies in width (1–3 feet), in depth and speed of flow. The water is brackish and there are many relatively quiet pools with a free growth of algae and grasses which affords an ideal environment for the breeding of *A. albimanus*.

It is not always easy to find anopheline larvae in pools or streams that appear to be ideal breeding places. A search of small puddles such as those that form in cow tracks at the border of the pool may

be necessary. It is sometimes helpful to stir up the mud in a small pool so that the larvae, which have been hiding on the bottom, will come up to the surface where they can be easily spotted against the muddy background.

Isolated storm water pools were a troublesome source of pest mosquito breeding but in only one such pool were anopheles found. The explanation is believed to be that the storm water usually evaporates before a growth of algae develops which the anopheles appear to demand in their breeding places. These pools cannot be neglected, however, because of the indeterminate length of time they may persist. As mentioned before, rain-water pools here rapidly become brackish from the salt in the soil.

Low land that becomes marshy after heavy rains is a serious problem. The water is brackish, and the low grass growing through the shallow water provides the food and shelter, without barring the sunlight which *A. albimanus* requires (8). These temporary marshes constitute the most prolific anopheline breeding grounds in this vicinity.

For a time we were inclined to dismiss crab holes as possible anopheline breeding sites, though psorophora and aedes species were repeatedly found. Eventually, however, *Anopheles albimanus* was found in a crab hole in water that showed two-thirds as much salt as the water of the bay. In connection with crab holes, we were interested to find unidentified syphonate larvae in underground crab tunnels. The tunnels were opened with shovels, and not only larvae but adult mosquitoes were found 10 inches below the surface.

Mangrove swamps are present in a fringe around the greater part of the shore line, between the approximate limits of high and low tide. The maximum tidal variation is given as 14 inches, and as little as 6 inches during some phases. The thickets thus occupy flat stretches of varying widths around the periphery of the bay and the roots are awash at high tide. In seasons of lowest tide there is a period of 1 to 2 weeks when parts of the swamps do not receive the daily tidal flooding. If, during this period salt water trapped in the swamp is diluted by fresh water from rains, conditions favorable to the breeding of *A. albimanus* obtain. *Albimanus* is reported to breed in mangrove swamps in some parts of Cuba but none were found locally.

Engineering and construction projects on an expanding station produce a constantly changing topography with coincident interference with drainage and consequent collections of both waste water from the building operations and storm water. We have found occasional anophelines and great number of other larvae in such man-made pools.

Apparently *A. albimanus* does not favor tin cans, bottles, etc., as

breeding places; at least we found no larvae in such places during repeated searches. Neither did we find larvae in Lyster bags or water tanks.

Bromelaid plants, locally called "tree pineapples" or "air plants" are quite plentiful and infest a large proportion of the trees in many areas. These plants hold water well and it is not uncommon to find one or two cupfuls in a single one. It would seem easily possible for larvae to develop in bromelias, but we did not find them there.

The great majority of the mosquitoes found were of the genera *Culex*, *Aedes*, and *Psorophora*. *Aedes taeniorhynchus* and *Psorophora columbiana* are very common and are persistent biters. The chief anopheline species found was *albimanus* although rarely *A. crucians* was identified. *A. albimanus* is reported to be the important malaria transmitter in Cuba (1) so we feel safe in assuming that this species is responsible for malaria here. Unfortunately for the simplification of anopheline control, we have found *albimanus* in almost all the major types of water collections, from crab holes to running streams. Control measures, therefore, must be applied to practically all of the water collections. Successful control measures over this wide an area should result in the control on pest mosquitoes as well as the anophelines.

The degree to which mosquito control is effective is measured by the density of the mosquito population. It is therefore desirable to make sample counts from time to time in the vicinity of the sleeping quarters and to compare the results. It is difficult to estimate the number of mosquitoes by the degree of annoyance they produce and especially is this so in the case of species that are not particularly vicious biters. Moreover, only the roughest idea of the concentration of anophelines can be gained through inquiry and inspection in the various portions of camp. In order to obtain this information a sufficient number of adult mosquitoes must be captured and identified. If anophelines are present in only small numbers, a large sample must be collected in order to find them, and the collection by hand of several hundred mosquitoes requires considerable time and effort.

To facilitate the collection of mosquitoes, a trap using animal bait has been devised and used in the Caribbean area for a number of years. The trap is made large enough to serve as a stable for a calf, a donkey, or a horse, which is used as the bait. It is screened around the upper portion and is mosquito tight except for one and one-half inch slots at the apices of V-shaped entrances along the sides (fig. 1). The trap should be portable. The one described by Magoon (9) is put together in sections with wing nuts and bolts so that it may be taken part for easy transportation, and thin galvanized iron sheets are used for the roofing and siding to reduce the weight. Our trap could not be dismantled and was made of wood rather than sheet metal because

the other materials were not available. It was less convenient to move than the collapsible trap, but it served its purpose quite satisfactorily.

The principle of operation is the same as that of a fly trap; in fact, a fair quantity of flies are caught along with the mosquitoes. The insects fly in through the entrance slots, and, after having had their blood meal, are unable to find their way out. *Anopheles albimanus* is said to make no choice between calves and humans, biting either with equal vigor (10). Since some other species of mosquitoes discriminate between man and animal the total catch of the trap will not be a true cross section of the total mosquito population. However, the number of any given species should be comparable to the number caught at another time or location, and will serve to indicate an increase or decrease of that species.

The trap is set up just beyond the living quarters on the side of the camp toward the mosquito breeding areas so that the mosquitoes, in search of a blood meal, are intercepted before reaching the men. Before sundown the animal is put into the trap with a bucket of water for his comfort and is left there until morning, when the mosquitoes are captured with a collecting tube and killed.

We used both calves and a donkey as bait. The donkey, being more docile, proved more satisfactory than the calves.

The catch in 1 night varied widely in numbers. Near the worst breeding area a catch of 300 mosquitoes, including 15 anophelines, was made. Within the camp, the usual catch has been under 10 mosquitoes, of which 0 to 3 were anophelines. The trap thus serves to indicate the areas where additional search for breeding places is most needed.

Mosquitoes are effectively controlled by attacking the aquatic stages of their life cycle. Depriving the larvae of their development medium by draining all collections of water is the most efficient method, but it is also relatively long and costly. When such a project is prohibitively expensive, or pending the time that a drainage system may be put into operation, other measures must be used.

A film of oil over the surface of the water destroys larvae by the poisoning effect of the volatile gases liberated and to some extent by a suffocating action in clogging the breathing tube. A suitable oil should spread rapidly and maintain a continuous thin film, volatilize sufficiently to poison the larvae but not evaporate too fast, and it should be inexpensive. Diesel oil answers these requirements. Used crankcase oil, diluted with one or two parts of kerosene, is satisfactory, though not as cheap or as convenient as diesel oil. The addition of crude castor oil is said to improve the spreading qualities of the mixture, but we did not have occasion to try this.

Oiling is most effective when used on relatively small bodies of



FIGURE 1.—ANIMAL-BAITED MOSQUITO TRAP.



FIGURE 2.—EQUIPMENT FOR THICK-FILM MALARIA SURVEY.

water. We were not successful in an attempt to oil a flooded athletic field, where the breeze carried all the oil to the leeward side, allowing larvae to mature in great numbers in other parts of the field. Oil is useful on running streams, as it tends to collect in the still pools where the larvae would grow. In treating a stream, the oil should be sprayed along the margins, where the current is least swift.

A number of methods for applying oil are available. The most generally useful is the knapsack sprayer, which is applicable to small pools, streams, and, to some extent, marshes. Drip cans may be used over small running streams, though in our hands this has not been as effective as spraying. Time may be saved in oiling large open pools by using a burlap drag, which is soaked with oil and dragged back and forth across the pool with lines. In constant pools of fair size we sank bags of oil-soaked sawdust. Oil may also be applied simply by pouring, though this method is rather wasteful.

Airplane dusting with paris green is effective against anopheline larvae but has no effect on larvae that do not feed on the surface. The large breeding areas here which might be treated by plane dusting are only temporarily marshy and are generally used as athletic fields and target ranges. Because of this it was considered hazardous to use arsenic; consequently no use was made of this method of control.

Cresol is an effective larvicide, and we used it in a limited number of situations where for some reason oil was not desirable. It must be stirred into the water to make a concentration of about 1/5000, in order to kill the larvae that are present; its effect does not persist.

Mosquito larvae may be markedly reduced by stocking the breeding pool with minnows that feed on larvae. A number of small fish have been found effective for this purpose, particularly *Gambusia affinis*. We did not attempt to use this method of control. However, we did note the presence of minnows in one shallow brackish pool that covered about one-half acre. No larvae were seen in this pool, although they were regularly found in adjacent puddles where minnows were absent.

Drainage is the permanent answer to the mosquito problem, if it is practicable. In many localities the expense of installing adequate drainage would be prohibitive; in others the expense is small enough to be well justified. Any such project, however, should be carefully studied before it is undertaken, and it is not impossible that an improperly constructed drainage system might increase rather than decrease mosquito breeding areas. In order to facilitate drainage of storm water from the low ground near camp we dug shallow ditches. As mentioned earlier, the camp was located on a series of peninsulas, between which lie flats that are inundated by freshets after heavy rains, with resulting persistent pools. In one such area we cleaned

out and enlarged the stream beds in an effort to confine the storm water to the stream courses. This work was not permanent in character, and it was anticipated that each rain would result in a partial filling up with silt of the enlarged water courses. A permanent drainage project would probably involve concrete lined ditches similar to those recommended and used by the Rockefeller Foundation in Cuba.

Clearing the underbrush as a measure of malaria control must be carefully weighed, as the major vector in Cuba, *A. albimanus*, prefers breeding pools that are exposed to sunlight. Therefore, when an area is cleared, drainage and oiling must be employed to prevent mosquito breeding; otherwise pest mosquitoes that breed in the shaded pools may be replaced by *A. albimanus* when the pools are left open to the sun. With due consideration for this factor, clearing the thick undergrowth in the vicinity of camp was undertaken with a view to eliminating daytime sanctuaries of adult mosquitoes and making the pools more accessible for drainage and oiling.

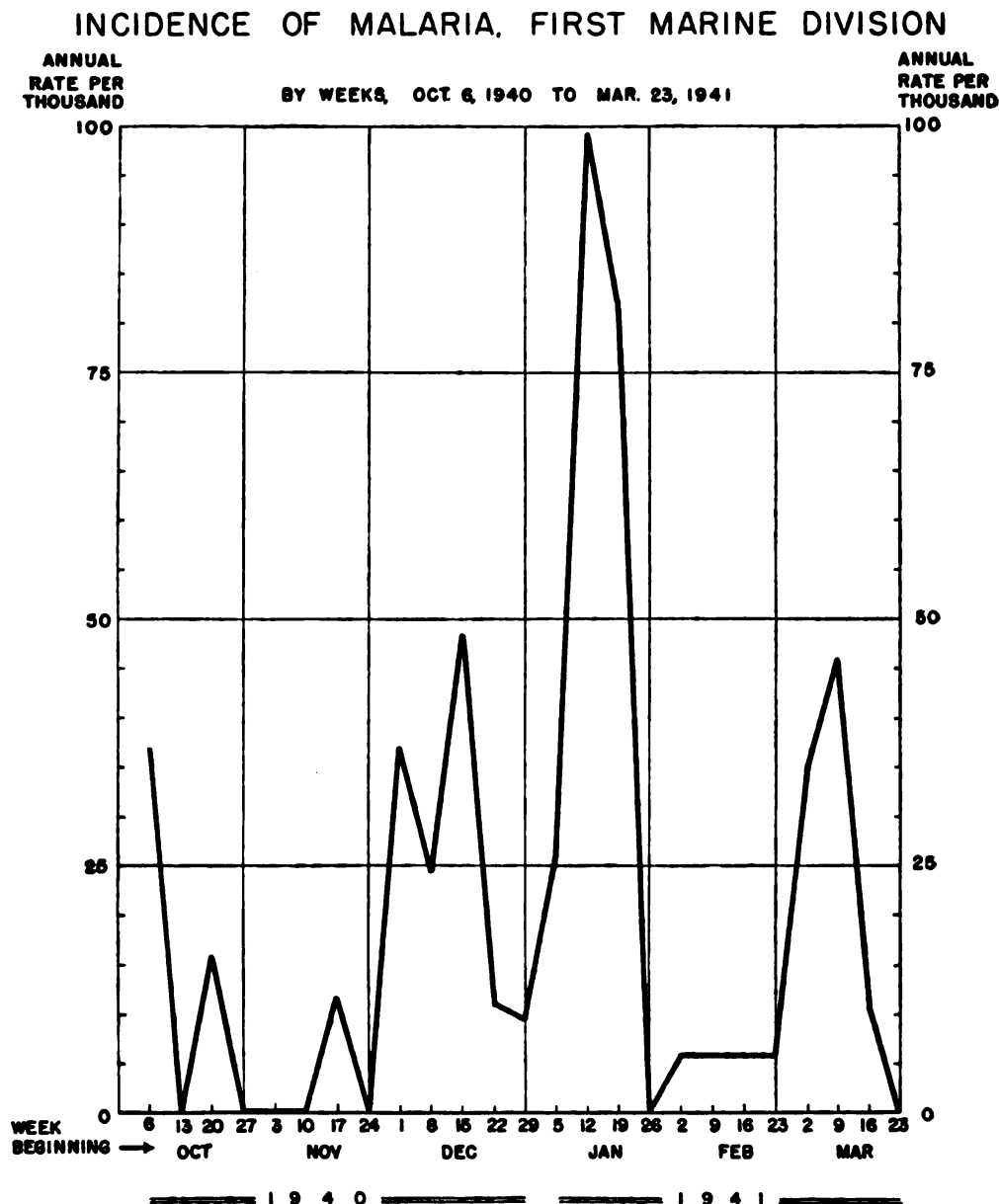
MEASURES FOR THE DISCOVERY AND TREATMENT OF INFECTED INDIVIDUALS

Personnel of the division who developed malaria were promptly transferred from the command to the Mobile Base Hospital, as soon as the diagnosis was made. There the usual malarial precautions were taken and treatment given. No man was returned to duty until his course of treatment was completed and his blood film negative for malarial parasites. All personnel admitted to the sick list, regardless of diagnosis, had malarial smears taken.

The first two cases of malaria to develop in the division were in the aircraft group and were considered to be recurrences, using James' (11) definition of recurrence as a return of fever and parasites at some time later than 24 weeks; a relapse as a return between 8 and 24 weeks, and a recrudescence as a return within 8 weeks of recovery from the primary attack.

During the month of December 10 more cases developed, which were equally divided among the troops who had been away about a month on maneuvers and the 600 who had remained in camp. At this time sporadic cases developed among the personnel at the naval station. The incidence of malaria in the division from the time of our arrival until the time of this report is shown on table II. The same thing is shown graphically in figure 3. There were 61 admissions to the sick list of which 28 were definitely attributed to this area; 14 apparently contracted the infection elsewhere; in 13 it was impossible definitely to state whether the infection was acquired here or elsewhere; and in 6 cases the admission was because of a return of symptoms following a previous infection. Forty-three of these 61 cases were of the benign tertian type, 16 were malignant tertian, 1 was a mixed infection—benign and malignant tertian—and in 1 the type wasn't determined.

the diagnosis being made on a single thick film. No quartan malaria was found. Two of our 16 cases of malignant tertian malaria died. In one, death occurred with cerebral symptoms; in the other, the symptoms were suggestive of blackwater fever.



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FIGURE 3.

The occurrence of these cases definitely attributable to this area among persons who had no previous history of malaria and who had not been off the station in over 2 months suggested the probability that our reservoir of infection might be the native workmen employed

by the contractor and living on the station. This supposition was further strengthened by the knowledge of endemic malaria in the neighboring towns from which these workmen came. Consequently actual case finding in this group by means of a thick-film survey was started the first of January in order that carriers might be detected and placed under treatment.

TABLE II.—*Incidence of malaria, first marine division*

Week beginning—	Number of cases	Annual rate per 1,000	Source of infection
Oct. 6, 1940.....	1	37	Recurrence.
Oct. 13, 1940.....	0	0	
Oct. 20, 1940.....	1	16	Do.
Oct. 27, 1940.....	0	0	
Nov. 3, 1940.....	0	0	
Nov. 10, 1940.....	0	0	
Nov. 17, 1940.....	1	12	Local.
Nov. 24, 1940.....	0	0	
Dec. 1, 1940.....	3	37	All local.
Dec. 8, 1940.....	2	24	Do.
Dec. 15, 1940.....	14	48	All elsewhere.
Dec. 22, 1940.....	1	11	Local.
Dec. 29, 1940.....	1	9	Do.
Jan. 5, 1941.....	3	26	Do.
Jan. 12, 1941.....	12	99	4 local; 1 elsewhere; 7 undetermined.
Jan. 19, 1941.....	12	82	7 local; 1 elsewhere; 4 undetermined.
Jan. 26, 1941.....	0	0	
Feb. 2, 1941.....	1	6	Recurrence.
Feb. 9, 1941.....	1	6	Undetermined.
Feb. 16, 1941.....	1	6	Elsewhere.
Feb. 23, 1941.....	1	6	Recurrence.
Mar. 2, 1941.....	16	35	2 local; 3 elsewhere; 1 relapse.
Mar. 9, 1941.....	8	46	3 local; 4 elsewhere; 1 undetermined.
Mar. 16, 1941.....	2	11	1 local; 1 recrudescence.
Mar. 23, 1941.....	0	0	

¹ 1 death.

THICK-FILM SURVEY

By the thick-film method four to five times as much blood as can be made into a satisfactory thin film can be examined and the searching time reduced from half an hour or more to 5 or 6 minutes. The thick film is confusing at first to one familiar with thin films stained with Wright's or other polychrome stains, but those who know the parasites on the thin film have little difficulty in quickly learning to detect them by this method.

For the survey, the work was divided among two groups—one to collect the blood specimens at the job where the men worked; the other to stain and examine the slides. Both groups were under the direction of a medical officer. The field group consisted of two technicians to make the films and a clerk who marked the slides with a number corresponding to the man's name or employment number, which was entered in the field book. The laboratory technicians stained and examined these slides the following morning, reporting them by number only, the result then being listed in the field book by the clerk.

No effort was made to determine the splenic index on this group, since all were adults. In malarial surveys on children, the splenic

index is of some value, but among adults its value is questionable. In an effort to determine the incidence of carriers among those who gave a history of having had malaria as compared to those giving no history of the disease, all were carefully questioned on this point. Of the 1,851 individuals examined, 269 gave a history of having had malaria anywhere from 2 months to 30 years previously, and in this group 15 carriers were identified—a rate of approximately 5 percent. Among the 1,582 giving no history of malaria, 31 carriers were identified—a rate of approximately 2 percent. Among the 701 marines of the group who were surveyed, 102 gave a history of having had malaria anywhere from 2 months to 20 years previously, and in this group 1 carrier was detected—a rate of 0.98 percent. One carrier was also found in the other 599 examined—a rate of 0.17 percent. We doubt the accuracy of the history among the native workmen, but the figures do show that having had recognizable clinical malaria, the chances of being a carrier are greatly enhanced.

Field equipment.—The field equipment needed depends upon the number of individuals to be examined each day and varies only in amount, not in character. Each morning before leaving, the field group places in a satchel, or other suitable carrier, sufficient clean glass slides, slide boxes with stands, and a jar filled with gauze squares soaked in 70 percent alcohol for the day's needs. In addition, blood lancets, a wax pencil for marking slides, the field book, and a lead pencil for entering data in the field book are needed (fig. 2).

On each patient we took two slides. A thin film was made on one and on the other one we made two thick-film preparations, one at each end of the slide. This was done for two reasons; first, if something should go wrong with the staining we had another specimen available without again looking up the man, and, second, where the thick drop was positive yet the type not definite, we had a thin smear to examine for type identification. Only in positive cases were the thin films stained. Occasionally this was of some help, but where the thick film showed only one or two parasites we were usually unable to pick up any in the thin smear.

The slides used were those issued by the Supply Depot. Survey slides were cleaned and reused until they became fogged and had to be discarded. We found that a slide could be used and cleaned about five times before it had to be discarded. In cleaning, the cedar oil and the stained film was removed as far as possible with a piece of gauze soaked in xylol after which they were washed in warm soapy water and placed overnight in 70 percent alcohol. The following day they were dried, replaced in the box in which they came and reissued for use.

The slide boxes were the usual two section type holding 100 slides. Since we took two slides on each case each box held 50 cases.

Blood film.—The thick-film at each end of the slide was made from a large drop of blood taken from the finger and placed about half an inch from the end of the slide. The placing of the drop in this position permits a full coverage of the stained film without the mechanical stage interfering with the objective. The drop is then worked into a circular spot about half an inch in diameter and placed in the slide box to protect it from dust while drying. It is important that the slide dry in a horizontal position, otherwise it tends to run, the outline becomes irregular, and much time is lost in following the protrusions during examination. In order to accomplish this the slide boxes are held in an upright position by rubber bands to a right-angled support made by nailing two boards together. When the box is filled it is closed to protect the slides from flies and dust.

Drying is the only thing causing the blood to adhere to the slide during staining. The time for this varies with the climate unless an incubator, which we did not have, is used. At incubator temperature the slide is sufficiently dry to stain in from 1 to 1½ hours. We found we got the most uniform results by letting the slides taken one day air dry overnight before staining.

For a blood-lancet we used a needle inserted into the cork stopper of a two ounce bottle filled with alcohol. When the cork is replaced the needle is submerged in the alcohol and thus made ready for the next case.

Staining.—In staining we followed the method of Barber and Komp (3) using the modification of Giemsa's stain recommended by Benavides (12) and found it entirely satisfactory. This stain does not require the use of Azur II. It is made up as follows: 2.4 grams of Azur II eosin is placed in a thoroughly cleansed Erlenmeyer flask and 200 cc. of C. P. anhydrous glycerin is added. The flask is stoppered with a well-fitted cork and then placed in a water-bath at 60° C. for 30 minutes. The flask is shaken once or twice during the period following which it is removed from the bath and 200 cc. of C. P. methyl alcohol added. After mixing the contents the flask is again placed in the hot-water bath for another 30 minutes, during which period it is shaken once or twice. It is then removed and placed in the incubator overnight at 37° C. It is then removed, passed through filter paper and stored in the dark in well stoppered 200 cc. bottles which have been thoroughly cleansed, washed with methyl alcohol, dried, and covered with tin foil and paper. This is the stock solution and in its preparation everything must be scrupulously clean.

Application of the stain requires dilution of this stock solution with water which is neutral or slightly alkaline (pH7.0 to pH7.2). We used freshly distilled water and found it satisfactory without correction. Benavides dilutes his stain in a proportion of 1 cc. to 1 ounce

of water and after thorough mixing leaves his slides in the staining solution for $1\frac{1}{2}$ hours. We used a dilution of 1:15 and stained our slides for 30 minutes with satisfactory results. Owing to a shortage of stain we saved this diluted stain in a bottle cleansed in the same manner as the storage bottles for the stock stain and reused it several times. As it got older it lost its strength and a longer staining period was required. When more than 40 minutes were required to satisfactorily stain a film the stain was discarded.

The slides with the thoroughly dried blood films were removed from the slide box and stained in batches of 25 by the simple method of putting pieces of cardboard 1 inch square and $\frac{1}{8}$ of an inch thick between the slides at the end carrying the identifying number, and binding them together with a rubber band in groups of five. These cardboard separators provide a staining space between the slides. Not having staining jars, we placed 5 groups of 5 slides each in a 250 cc. beaker containing a sufficient quantity of the stain to just cover the film. With staining jars, blocks of 25 or more slides can be satisfactorily stained by this method. At the end of the staining period the slides were removed from the stain and gently dipped in two changes of water like that used in diluting the stock stain and allowed to air dry by standing on a piece of blotting paper or on paper towels. The films must be thoroughly dry before they are examined.

As a result of this survey 46 carriers were identified among 1,851 natives on whom 3,495 thick-film studies were made. This gave us a carrier rate of 2.4 percent. One man was found positive on both the first and second surveys, but following a second course of treatment was negative on the third. Since every individual employed by the contractor and living on the station had a blood examination on each survey, the transitory nature of the labor population is shown by the fact that 783 had but one examination, 640 had two examinations, and only 428 were present for all three examinations.

TABLE III.—*Summary of 3 surveys on natives*

Total number of examinations.....	3,495
Total number of individuals examined.....	1,851
Individuals having 1 examination.....	783
Individuals having 2 examinations.....	640
Individuals having 3 examinations.....	428
Total number of positive cases.....	46
Percent of individuals with positive films (carriers).....	2.49

For the entire province of Oriente, Carr et al. (1) found a malarial incidence rate of 1 percent. It was not until we began our third survey, however, that we tried to correlate our carrier cases with the home town of the individual. In this way we learned that better than 90 percent of the workmen came from points in Oriente province and 50 percent of the total employed came from two cities in the province;

over 500 from Guantanamo City and a little under 100 from Santiago. Both of these cities have malarial rates far above that of the province as a whole; in Guantanamo City it is better than 10 percent, in Santiago better than 5 percent. With this background it is not unreasonable to assume that our rate would be higher. At the time of the third survey only 18 of the carriers were still working and of these 10 came from Guantanamo City, three from Santiago and three from other parts of Oriente. Only two cases came from points outside the province.

As carriers were identified they were reported to the station medical officer who had a chief pharmacist's mate locate the man and start him on treatment and supervised his treatment daily thereafter. The medicine for this treatment was supplied by the medical department of the station at no cost to the individual.

The treatment we used for the carriers consisted of quinine sulphate, grains 30, for 10 days, followed by plasmochin simplex $\frac{1}{3}$ grain, three times a day for 5 days. Atabrine was not used for two reasons; first we had no idea as to how large a group we would have to treat, and quinine is effective, less expensive, and our supply was greater; and second, the workmen were acquainted with quinine and took it readily. Further, many consider quinine the only safe remedy for the mass treatment of malaria. Dunn (13) recommends quinine combined with plasmochin in nontoxic doses for short periods with a view to reducing the relapse rate and attacking the sexual phase of the parasite of malignant tertian fever when it is present.

The amount of plasmochin, a highly toxic drug, which we gave, and the length of the course is somewhat more than that usually recommended but we were particularly anxious to attack the sexual phase of malignant tertian malaria. It is our feeling that persons taking plasmochin, regardless of the size of the dose, should be under close medical supervision in order that medication can be stopped with the development of the first symptoms of toxicity. This supervision we had and, fortunately, no ill effects of any kind were noted and no man missed a single day of work as a result of treatment. Individuals who were present for one or more thick-film studies after completing treatment, with one exception, showed a negative film on further examinations.

SUMMARY

1. Malaria is endemic in the area studied and is now a definite problem due to the importation of native laborers who are carriers.
2. All malaria in this area, whether active clinical cases or carriers, was of the benign and malignant tertian types. No cases of quartan malaria were found.
3. *A. albimanus* is reported to be the chief vector of malaria and

was found to be the most common type of anopheline on this station.

4. The control of mosquitoes at this station resolves itself, primarily, into a drainage control project.

5. The treatment of carriers identified through thick-film surveys, combined with measures to reduce the number of mosquitoes was accompanied by a lowering of the malarial incidence rate in the Division.

6. An animal baited mosquito trap as used in other areas of the Caribbean for the determination of mosquito species present is described.

NOTE.—We wish to express our appreciation to the Division Surgeon, Commander W. T. Brown (MC), United States Navy, whose interest and cooperation made this study possible. Also we wish to acknowledge the cooperation of Captain L. W. Johnson (MC), United States Navy, who allowed us the use of the laboratory facilities at the United States Naval Mobile Base Hospital No. 1, and Commander F. W. Hooker (MC), United States Navy, who was responsible for the treatment of carriers when identified. Further, we wish to acknowledge the help rendered in one way or another by the following members of the Hospital Corps:

Chief Pharmacist's Mate J. P. D. Ducharme; Pharmacist's Mates, first class, B. H. John and C. I. McIntosh; Pharmacist's Mates, second class, R. C. Davis and D. C. Sylvester; Pharmacist's Mates, third class, W. D. Loftus and R. D. Williams, and Hospital Apprentices, first class, M. Brodnax and R. E. Hanna.

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WATER DISTILLATION IN POLLUTED HARBORS

By Commander Alanson L. Bryan, Medical Corps, United States Navy

The problem of distilling drinking water while ships are in a polluted harbor is one of great concern to naval medical officers. At one time all ships were forbidden to run their evaporators while in certain harbors. The problem of obtaining potable drinking water from water barges and from the beach is a major task especially during fleet concentrations. Meticulous care in operation of evaporators and checking of the end product is necessary before it can be released for human consumption. With this in mind the following recommendation was made to the engineering officer of a battleship in harbor:

It may be well to again caution the evaporator personnel of this ship, in view of the lack of thermostat temperature warning signal on first effect, that spill-over and priming must be guarded against and add that no difficulty should be experienced distilling sterile potable water provided that the greatest of care and meticulous observations are carried out to prevent said priming, spill-over, and contamination, and that each batch of water must be tested by the Medical Department Laboratory Technician before it is released for ship's use.

It is noted that mention is made of the thermostatic controls on evaporators. Some ships are not equipped with this device. It is simply a thermostat that causes a bell to ring if the temperature of the first effect drops below a certain point.

The problem of priming, spill-over, and contamination is ever present even under meticulous care. To guard against this each batch of water is tested by Dominick-Lauter media before being released for general distribution and human consumption.

The bacteriological test with Dominick-Lauter media is a colorimetric and fermentation test and can be readily performed aboard ship. It is a simple test and is performed as follows:

This is a quick, simple method and is considered superior to practically all other methods. The water must be collected in a sterile container under sterile conditions. The media consists of 2 varieties, strong and weak. Use 2 tubes of each. Mix water well. Into each tube of the weak media pipette 1 cc. of the water; into each tube of the strong media pipette 10 cc. of the water. Incubate at 37° C. The test is read after 18 or 24 hours. A greenish-yellow discoloration of the media with gas formation is positive. The color change without gas, and gas without color change is not considered positive. The advantage of this test is that only organisms of the coli-aerogenes group of fecal origin will give a positive reaction. Closely related organisms of the nonfecal group will not give a positive reaction. By the use of the following chart one may arrive at a fairly accurate estimate of the number of coli-aerogenes organisms in 100 cc. of the water.

Positive with—		
10 cc. of water	1 cc. of water	Number of organisms per 100 cc. of water
0	0	0
0	1	5
0	2	10
1	0	7
1	1	12
1	2	20
2	0	15
2	1	25
2	2	33 +

E. g., if one tube of strong media (with 10 cc. of water) is positive and all the other tubes are negative the number of C. A. organisms present per 100 cc. is 7; if 1 strong and 1 weak are positive the number of organisms is 12 etc.

NOTE.—The media for the Dominick-Lauter test may be obtained dehydrated from the Difco Laboratories, Detroit, Mich. For the weak media dissolve 19.5 grams of the media in 1,000 cc. of distilled water. For the strong media dissolve 32.5 grams of the media in 1,000 cc. of distilled water. The fermentation tubes are prepared in the usual manner by inserting a small inverted test tube inside a larger test tube. Place 25 cc. of dilute media into each test tube of the dilute group and 25 cc. of strong media into each test tube of the strong group. Sterilize in the autoclave under 15 pounds pressure for 25 minutes.

The media is carefully prepared. The pH is tested and the media is cultured.

Color change and gas formation are both essential for a positive test, either one alone is not considered positive. The colon bacilli whose natural habitat is the intestines of man and many other animals is taken as an index of pollution. This test is designed to show the presence of colon bacilli, the absence of colon bacilli in water proves its harmlessness so far as bacteriology can prove it. Because it is fair to assume that typhoid bacilli, dysentery bacilli, and other intestinal parasites would not likely be present in water in the absence of colon bacillus.

The personnel that operate the evaporators are saline conscious and they have the layman's belief that water that is evaporated and contains only $\frac{3}{8}$ of a grain of salt per gallon is good enough for anyone to drink and they did not believe that we would ever obtain a bacteriological growth from such fine water. On February 16, 1941, we obtained a positive Dominick-Lauter test in water that had been made and put in tank C-3. This test was repeated and again found positive. The water was held in this tank and used for boiler feed water; it contained only $\frac{3}{8}$ of a grain of salt per gallon. This tank will not be used for stowage of potable drinking water until after it has been cleansed and painted.

Occasional water specimens and all positive specimens are sent to the Naval Hospital for checking. It is not practicable to send all

water specimens to the Naval Hospital for testing as they are already overburdened.

RÉSUMÉ

1. The Dominick-Lauter test can readily be performed aboard ship.
2. The test can be performed without the use of an incubator. However, we use an incubator and recommend its use if available.
3. It is not practicable nor is it necessary to use the old bacteriological methods of testing water aboard ship. If bacillus coli is present we condemn the water because other pathological organisms may be present.
4. Dominick-Lauter media should be made available to all large ships. We were unable to obtain it from any source in Honolulu.
5. This procedure is not considered a panacea for all water problems, however it gives invaluable information if properly carried out.

STATISTICS

HEALTH OF THE NAVY

The statistics (annual rates per 1,000) appearing in this summary were compiled from data contained in monthly reports of communicable diseases received in the Bureau for the months of January, February, and March 1941.

ENTIRE NAVY

Year	All diseases	Injuries and poisonings	All causes	Communicable diseases		Venereal diseases
				A	B	
1936.....	337	49	386	30	140	42
1937.....	276	36	313	18	98	59
1938.....	387	46	434	9	128	86
1939.....	423	47	470	19	168	80
1940.....	499	49	548	21	206	90
1941.....	591	45	636	87	254	62

FORCES ASHORE

1936.....	518	50	568	59	226	26
1937.....	312	36	347	34	131	21
1938.....	447	50	497	14	195	40
1939.....	487	47	534	10	253	41
1940.....	583	43	626	26	300	49
1941.....	759	46	805	140	370	42

FORCES AFLOAT

1936.....	229	49	278	13	89	51
1937.....	256	37	293	8	78	78
1938.....	353	44	397	6	90	113
1939.....	390	47	437	24	125	100
1940.....	439	54	493	17	139	119
1941.....	458	44	502	46	163	78

Common infectious diseases of the respiratory type.—A total of 18,880 admissions for these diseases was reported for the entire Navy for the first quarter of 1941, or a 105-percent increase when compared with the corresponding quarter in 1940. Catarrhal fever was responsible for 14,840 of the total admissions for respiratory diseases.

There were 12,285 admissions for these diseases reported by shore stations in the United States, 6,454 from forces afloat, and 141 from

outlying naval stations. The largest number of cases were reported from the following ships and stations:

Ship or station	January	February	March	Total
Naval Training Station, Great Lakes, Ill.....	795	702	847	2, 344
Naval Training Station, Newport, R. I.....	409	431	418	1, 258
Naval Training Station, Norfolk, Va.....	350	300	199	849
1st Marine Division, F. M. F., Guantanamo Bay, Cuba.....	428	225	(¹)	653
Marine Barracks, Quantico, Va.....	404	71	100	575
Marine Corps Base, San Diego, Calif.....	201	174	195	570
Naval Air Station, Pensacola, Fla.....	373	110	60	543
Marine Barracks, Parris Island, S. C.....	220	45	29	294
Naval Air Station, Jacksonville, Fla.....	64	77	122	263
Naval Air Station, Norfolk, Va.....	119	72	62	253
Naval Academy (midshipmen), Annapolis, Md.....	204	6	15	225
Naval Dispensary, Washington, D. C.....	117	65	29	211
4th Defense Battalion, F. M. F., Guantanamo Bay, Cuba.....	155	28	19	202
Norfolk Navy Yard, Portsmouth, Va.....	81	66	43	190
Naval Training Station, San Diego, Calif.....	89	24	73	186
Receiving Ship, New York, N. Y.....	64	71	50	185
U. S. S. <i>Prairie</i>	79	54	42	175
Naval Air Station, San Diego, Calif.....	65	59	46	170
5th Defense Battalion, F. M. F., Parris Island, S. C.....	133	26	5	164
Submarine Base, New London, Conn.....	52	65	45	162
U. S. S. <i>California</i>	51	54	37	142
1st Marine Division, F. M. F., Quantico, Va.....	(²)	(²)	142	142
U. S. S. <i>Saratoga</i>	77	50	13	140
U. S. S. <i>Enterprise</i>	71	52	13	136
Navy Yard, Philadelphia, Pa.....	65	36	35	136
U. S. S. <i>Lexington</i>	60	40	31	131
U. S. S. <i>Colorado</i>	67	38	20	125
Battle Force, Fleet Air Detachment.....	39	47	35	121
U. S. S. <i>Arizona</i>	46	32	41	119
Naval Reserve Signal School, Chicago, Ill.....	62	19	26	107
U. S. S. <i>Arkansas</i>	73	21	10	104
Navy Yard, Charleston, S. C.....	58	27	16	101
U. S. S. <i>Louisville</i>	16	61	23	100
Naval Reserve Aviation Base, Squantum, Mass.....	69	17	14	100

¹ See 1st Marine Division, F. M. F., Quantico, Va.

² See 1st Marine Division, F. M. F., Guantanamo Bay, Cuba.

In reporting the occurrence of respiratory diseases at various stations, the senior medical officers commented as follows:

Naval Academy, Annapolis, Md. (Sanitary report for January 1941).—There was quite an increase in catarrhal fever, acute, cases admitted to the excused and sick list during the month. All cases with a temperature of 99° F. or above were admitted to the sick list. The cases were extremely mild and without complications.

Marine Barracks, Washington, D. C. (Sanitary report for January 1941).—During the month there was a total of 73 admissions for communicable diseases as follows: 4 for tonsillitis, acute; 1 for laryngitis; and 68 for catarrhal fever, acute. The cases of catarrhal fever were

discharged to duty after two days on the sick list. In 11 cases transferred to hospital the diagnosis was changed to influenza.

Naval Training Station, Newport, R. I. (Sanitary report for February 1941).—Catarrhal fever, acute, has apparently been endemic and epidemic in localities in the New England States. The 335 cases reported does not appear an excessive percentage under these circumstances. The infection in general has been mild in character.

Naval Ammunition Depot, Hingham, Mass. (Sanitary report for January 1941).—There was a mild epidemic of catarrhal fever, acute, on the station. There were 21 admissions for service personnel, 30 cases among civil-service employees, and 10 cases among dependents.

Naval Air Station, Quonset Point, R. I. (Sanitary report for January 1941).—There was a minor epidemic of catarrhal fever, acute, during the month, 28 cases being admitted to the sick list. Influenza has been prevalent in this locality.

Naval Air Station, Lakehurst, N. J. (Sanitary report for January 1941).—Sixteen cases of catarrhal fever, acute, developed during the month incident to the marked change to colder weather with considerable snow and low temperature. The disease began with a fever, general malaise, headache, nasal obstruction, and some coughing. Some of the symptoms were suggestive of mild forms of influenza. Cases were discharged to duty after 2 or 3 days on the sick list. There were no complications.

Naval Air Station, Pensacola, Fla. (Sanitary reports for January, February, and March 1941).—There were 373 admissions to the sick list for diseases of the upper respiratory tract during the month of January—365 cases of catarrhal fever, acute; 2 cases of influenza; and 6 cases of tonsillitis, acute. The diseases were mild and without complications.

There were 107 admissions to the sick list for diseases of the upper respiratory tract during the month of February—97 cases of catarrhal fever, acute; 1 case of influenza; 6 cases of tonsillitis, acute; and 3 cases of pharyngitis, acute. The diseases continued mild and without complications.

There were 60 admissions to the sick list for diseases of the upper respiratory tract during the month of March—49 cases of catarrhal fever, acute; and 11 cases of tonsillitis, acute.

Naval Air Station, Jacksonville, Fla. (Sanitary reports for the months of January and February, 1941).—During the month of January there was prevalent at this station a mild form of influenza in addition to the seasonal incidence of acute catarrhal fever. While these attacks of influenza, in the main, have been of a mild type, 2 of the 28 cases admitted to the sick list were cases complicated by bronchial pneumonia. There were 39 admissions for acute catarrhal fever.

Influenza and catarrhal fever continued to be prevalent at this Station during the month of February.

Fourth Defense Battalion, F. M. F., Guantanamo Bay, Cuba (Sanitary report for January 1941).—The health of the command has been good except for a sharp increase in the admissions for catarrhal fever, acute, and mild influenza. The average duration of illness was from 2 to 4 days. No cases were seriously ill.

Naval Ammunition Depot, Iona Island, N. Y. (Special report dated January 20, 1941).—Sixteen cases of influenza occurred among the 88 marines stationed at this station between January 11 and 19. The cases were of a mild nature, were temperature-free within 24 to 48 hours, and required 1 to 4 days convalescence to regain strength. The symptoms noted were headache, body aches and chills, and pharyngeal irritation. Admission temperatures, 101° F. to 103° F. Laboratory findings showed normal urinalysis and white blood counts varying from 5,000 to 9,800.

Overcrowding of marine barracks and the prevalence of influenza throughout New York and neighboring states were believed to be the cause of this outbreak. The usual steps to prevent spread were carried out.

In addition to the 16 admissions for influenza there were 2 admissions for catarrhal fever and 1 admission for acute tonsillitis.

Dispensary, 11th Naval District, San Diego, Calif. (Sanitary report for January 1941).—A recurrence of mild influenza was noted among the personnel of the Eleventh Naval District Headquarters and United States Naval Supply Department. However, it was not true to form as one of the primary symptoms was a pharyngitis of varying intensity followed by typical malaise.

Naval Air Station, Sitka, Alaska (Sanitary report for January 1941).—The general health of the personnel has been excellent except for an epidemic of influenza. The onset of the epidemic occurred in December, and during the first half of January there were 30 cases admitted to the sick list. They were mild in character, uncomplicated, and averaged 3 sick days per case.

U. S. S. McCawley (Special report dated February 9, 1941).—There were 133 cases of catarrhal fever, acute, on board during the month of January involving marine-troop passengers and personnel of the crew. It is believed that the outbreak can be traced to unseasonable weather in the Guantanamo area during the week of January 9, just prior to the embarking of marines on board this ship, and to overcrowded living compartments after embarkation.

The average case complained of fever, ranging from 100° F. to 104° F., chills, and general body aches. The temperature was generally diphasic. The average duration of each case was 3 days; no complications were encountered.

U. S. S. Boise. (Special report dated March 21, 1941).—From February 23 to March 6, inclusive, 20 cases of tonsillitis, acute, and 4 cases of pharyngitis, acute, were admitted to the sick list.

Both tonsillitis and pharyngitis cases are included in this report because the symptoms were the same and the same type organism was noted in smears from all cases.

On February 26, eight cases of tonsillitis, acute, were admitted and it was evident that an epidemic was in progress. Ten cases were admitted the next day, four of which were classified as pharyngitis, acute, as the patients had had tonsillectomies. The epidemic ceased very abruptly, only three cases being admitted in the next 7 days. No cases occurred among officers, warrant officers, or chief petty officers.

The most prominent complaint was "sore throat." In a few cases the soreness was only mild but most patients stated there was considerable pain on swallowing. Most cases complained of generalized aching in muscles and joints. A few had chills. Admission temperatures ranged from 99° F. to 104° F., averaging 101° F. The tonsils in most cases were moderately enlarged, cryptic, acutely inflamed, and studied with greyish-white spots. No definite membrane was seen in any case. In some cases the lymphoid tissue of posterior pharyngeal wall was involved. The pseudo-membrane on tonsils and posterior pharyngeal wall could be wiped away without bleeding. Smear from the exudate revealed short chain streptococci in all cases. Definite improvement was noted in 48 hours.

From February 21 to 25 this vessel was in drydock. Men working under somewhat adverse conditions, due to rain, probably sustained sufficient lowered resistance which rendered them more susceptible when exposed.

The setting of material condition Baker and Affirm, causing some interference with proper ventilation, does not seem to be a factor in this epidemic because 18 of the 24 cases were admitted within 48 hours after leaving Pearl Harbor. Exposure undoubtedly occurred while the ship was at anchor in Pearl Harbor or in drydock. Practically all divisions were represented. The largest number from any one division was five.

The cafeteria system of feeding is used on this vessel which results in far less handling of food and insures protection from mass infections. The dishwashing machines are comparatively new and operate satisfactorily.

Control and other measures used are listed as follows:

(a) Immediate inspection of food handlers, barbers, and ship's store and soda-fountain personnel.

(b) Word passed for all men with any degree of sore throat to report for examination.

(c) Search for and elimination of all common drinking cups.

(d) Flaming of all scuttle butts three times a day and instruction in proper use of scuttle butts. (Some men were seen to touch their lips and mouth to fountain while drinking.)

(e) Checking on operation of dishwashing machines.

(f) Check up of "head to foot" sleeping arrangements.

(g) Individual paper cups were issued frequently to each patient.

(h) Sputum cups were issued.

(i) Mess gear was washed thoroughly with soap and soaked in 2% cresol solution before being returned to scullery for final sterilization.

The usual routine and symptomatic treatment, consisting of antiseptic mouth wash, salicylates, and Dover's powders, was given.

Ten of the 20 cases of tonsillitis, acute, received sulfanilamide over a 3-day period, total dosage averaging 180 grains! No toxic manifestations were noted. Sulfanilamide did not appear to hasten recovery. The cases which did not receive sulfanilamide seemed to recover as quickly as the cases which received the drug. This fact may have been due to the low virulence of the etiological organism. No complications were noted.

Other infectious diseases.—There were 695 admissions for measles, 3,198 admissions for German measles, and 665 admissions for mumps during the first quarter of 1941. Ships and shore stations reporting the greatest number of admissions were as follows:

Ship or station	Measles	German measles	Mumps
U. S. S. <i>Arizona</i>	0	21	28
U. S. S. <i>Arkansas</i>	0	33	1
Battle Force Fleet Air Detachment.....	3	29	0
U. S. S. <i>Boise</i>	0	25	0
U. S. S. <i>California</i>	24	3	0
U. S. S. <i>Chesler</i>	0	22	0
U. S. S. <i>Chicago</i>	4	17	7
U. S. S. <i>Colorado</i>	1	38	0
U. S. S. <i>Enterprise</i>	3	47	0
U. S. S. <i>Helena</i>	2	11	0
U. S. S. <i>Heywood</i>	0	24	0
U. S. S. <i>Idaho</i>	3	13	30
U. S. S. <i>Indianapolis</i>	2	24	0
U. S. S. <i>Lexington</i>	2	32	0
U. S. S. <i>Minneapolis</i>	0	23	0
U. S. S. <i>Mississippi</i>	0	16	0
U. S. S. <i>Nevada</i>	29	21	2
U. S. S. <i>Oklahoma</i>	0	33	9
U. S. S. <i>Phoenix</i>	0	4	7
U. S. S. <i>Quincy</i>	0	4	70
U. S. S. <i>Raleigh</i>	0	24	0
U. S. S. <i>Ranger</i>	25	0	1
U. S. S. <i>Salt Lake City</i>	0	44	0

Ship or station	Measles	German measles	Mumps
U. S. S. <i>Saratoga</i>	11	100	0
U. S. S. <i>Savannah</i>	0	23	0
U. S. S. <i>Tennessee</i>	1	25	0
U. S. S. <i>Vincennes</i>	0	1	12
U. S. S. <i>West Virginia</i>	0	22	0
U. S. S. <i>Yorktown</i>	1	24	1
Naval Training Station, Newport, R. I.....	7	249	0
Submarine Base, New London, Conn.....	2	11	7
Receiving Ship, New York, N. Y.....	15	3	0
Navy Yard, Philadelphia, Pa.....	5	5	3
Naval Academy (midshipmen), Annapolis, Md.....	5	63	62
Naval Academy (others), Annapolis, Md.....	0	3	52
Marine Barracks, Quantico, Va.....	2	107	0
Naval Air Station, Norfolk, Va.....	4	25	20
Naval Training Station, Norfolk, Va.....	169	162	153
Norfolk Navy Yard, Portsmouth, Va.....	7	37	9
Naval Air Station, Jacksonville, Fla.....	1	74	6
Naval Air Station, Pensacola, Fla.....	3	126	7
Naval Reserve Midshipmen School, Northwestern University, Chicago, Ill.....	18	83	0
Naval Training Station, Great Lakes, Ill.....	27	209	5
Marine Corps Base, San Diego, Calif.....	31	331	25
Naval Air Station, San Diego, Calif.....	4	58	1
Hospital Corps School, San Diego, Calif.....	3	20	1
Receiving Station, San Diego, Calif.....	1	58	1
Naval Training Station, San Diego, Calif.....	69	158	14
Navy Yard, Mare Island, Calif.....	13	53	0
Puget Sound Navy Yard, Bremerton, Wash.....	0	65	0

Measles—5th Defense Battalion, F. M. F., Marine Barracks, Parris Island, S. C.—(Sanitary report for February 1941).—There were 10 admissions for measles during February 1941. The admissions occurred within a period of approximately 60 hours and were confined exclusively to food handlers and men who prepared and served the general mess. Due to these pertinent facts a general epidemic of measles was expected and special precautions were instituted.

The entire battalion was mustered and individually examined by a medical officer; particular attention was given to the examination of respiratory tract and cutaneous system for evidence of erythematous rash; nose and throat sprays were given daily; and all messmen and cooks were inspected daily.

German measles—U. S. S. *Boise*—(Special report dated March 12, 1941).—From January 26 to February 28 there were 24 cases of German measles admitted to the sick list. There were no complications. The exact source of the infection was not definitely determined. The cases showed typical signs and symptoms, such as enlargement of lymph nodes, chiefly the posterior cervical chain, and rash. Examination of oral mucous membrane failed to reveal any Koplik's Spots. Admission temperatures ranged from 99° F. to 102° F. Isolation and routine precautions against spread of the disease were instituted. Cases averaged 5 days on the sick list.

Fifth Defense Battalion, FMF, Marine Barracks, Parris Island, S. C.—(Sanitary report for March 1941).—During the first 2 weeks of March an epidemic of German measles occurred and 25 cases were admitted to the sick list. A working quarantine was instituted and men from this Battalion were advised against attending public gatherings and movies.

Mumps—U. S. S. Quincy—(Special report dated April 9, 1941).—A case of mumps appeared on board on December 24, 1940, while this vessel was undergoing overhaul in the Norfolk Navy Yard. The patient had been home on leave. A second case appeared on January 15, 1941; 3 more cases on January 27; 21 cases from January 28 to February 2; and 14 cases from February 3 to February 5. Cases continued in varying numbers until March 21. All of the cases were transferred to hospital.

A record of contacts was begun on January 12, and an attempt was made to examine these persons daily at the sick bay. The number of contacts attained such proportions as to make this type of examination impracticable after February 4, following which date the men were examined at quarters under the supervision of division officers, who were instructed in the recognition of mumps. Possible cases were sent to the sick bay for further examination.

Head to foot sleeping arrangements of the crew had been in effect long before the appearance of the epidemic, and this policy, together with frequent inspections, was continued.

Although the ship was often in Condition Y, there was no restriction of ventilation. The possibility of spreading the epidemic by such restriction was realized.

Frequent inspections of the scullery and the operation and mechanical efficiency of the dishwashing machine were carried out. Trays were given a preliminary washing in soapy water and creosote before being placed in the machine. Other articles were further sterilized in boiling water after being run through the machine. No dish towels were used.

A possible source of contact between the men contributing to the spread of respiratory diseases was present in unauthorized "coffee messes." The sanitary hazard consisted in the habit of placing used cups together in a pail of water. Since the drinking of coffee is difficult to regulate, the sanitary hazard was best remedied by the use of paper cups.

The men were separated as much as possible by having men who had not had mumps sleep top side.

Frequent inspections of drinking fountains were carried out to see that the stream of water was of sufficient force so that men did not have to touch lip to spout.

Isolation aboard ship is difficult, but was attempted by the use of sheets.

The ordinary accepted methods of treatment were used. The sulfanilamide derivatives were used on a few cases and intramuscular injections of 35 cc. of immune serum was used in one case without beneficial results.

Because of the danger of starting a mumps epidemic throughout the Fleet, transfers of personnel to other ships were withheld until February 12, when permission from CinClant was obtained to transfer men who had previously had mumps. Ships receiving these men were instructed to take appropriate precautions. On March 15 the epidemic had subsided and authority was granted to transfer men without reservation.

Opinion as to cause and spread of epidemic.—The prevalence of diseases of the upper respiratory tract among the personnel was thought to have caused a general lowering of resistance and an increased susceptibility to mumps.

Overcrowded conditions aboard ship.

The presence aboard ship of 200 recruits with only 6 or 7 weeks of service.

The contamination of ship's water supply was not considered a likely possibility. Examination was made for possible contamination of fresh water tanks by cross connection with sewage line.

The possibility that wholesale distribution of the causative virus may have occurred through the scullery was also considered. Until a dishwashing machine is invented which will not operate at all unless it is operating correctly, the possibility of spread of communicable diseases through the scullery exists.

Statistical information

1. Total cases of mumps in epidemic.....	75
2. Percent of crew acquiring disease.....	8
3. Percent of crew giving previous history of mumps.....	55
4. Percent of crew giving previous history of mumps who acquired disease a second time in this epidemic.....	10.6
5. Average age of patients.....	25
6. Percent of 6 months' recruits aboard.....	22
7. Percent of 6 months' recruits aboard and in the division giving the highest percentage of cases.....	63
8. Percent by ratings:	
AS.....	18
S2c.....	24
S1c.....	24
P. O., 3c.....	9
P. O., 2c.....	9
P. O., 1c.....	8
C. P. O.....	4
Officers.....	4
9. Five percent of "Mumps Contact List," consisting of 120 men, acquired mumps as compared to 8 percent of crew as a whole.	
10. Average days on sick list.....	12
11. Percent of orchitis cases.....	31

The following observations were recorded.—1. Given certain conditions as regards season of the year, presence aboard of numbers of recruits, overcrowding, etc., mumps may assume considerable importance on a ship of the Navy.

2. Examination of contacts is more useful for the publicity given to the disease, with consequent voluntary reporting of cases, rather than to discovery of cases at time of inspection.

3. Upon the appearance of the first case it is important to obtain names of men who have previously had the disease, in order that replies obtained at a subsequent time will not be influenced by personal prejudices.

4. Difficulty in properly isolating cases was experienced due to inadequate space. Ships of this type require hospital facilities when a disease of this kind appears in epidemic form.

Chickenpox.—There were 30 cases of chickenpox reported during the quarter as follows:

Ship or station	January	February	March	Total
U. S. S. <i>Ballard</i>	0	0	1	1
U. S. S. <i>Colorado</i>	1	0	0	1
Inshore patrol, Norfolk, Va.....	1	0	0	1
U. S. S. <i>Livermore</i>	1	0	0	1
U. S. S. <i>Philadelphia</i>	0	1	2	3
U. S. S. <i>Phoenix</i>	0	0	1	1
U. S. S. <i>Salt Lake City</i>	0	0	1	1
U. S. S. <i>Seminole</i>	1	0	0	1
U. S. S. <i>St. Louis</i>	0	0	1	1
Submarine Base, New London, Conn.....	1	0	0	1
District Headquarters, New York, N. Y.....	0	1	0	1
Receiving Ship, New York, N. Y.....	0	0	1	1
Marine Barracks, Quantico, Va.....	1	1	0	2
Navy Yard, Washington, D. C.....	0	0	1	1
Naval Air Station, Norfolk, Va.....	0	0	1	1
Norfolk Naval Hospital, Portsmouth, Va.....	1	0	0	1
Post Dispensary, Marine Barracks, Parris Island, S. C.....	1	0	0	1
Naval Recruiting Station, Nashville, Tenn.....	1	0	0	1
Naval Training Station, Great Lakes, Ill.....	0	1	0	1
Naval Training Station, San Diego, Calif.....	0	2	0	2
Naval Air Station, Seattle, Wash.....	1	1	1	3
Navy Yard, Pearl Harbor, T. H.....	0	0	1	1
Naval Station, Guam, M. I.....	0	1	1	2

Septic sore throat.—Forty-nine cases of septic sore throat were reported for the quarter, 32 of which occurred at the Naval Reserve Radio School, Noroton, Conn. The remaining 17 cases were reported by 10 shore stations and 1 ship.

Scarlet fever.—There were 104 cases of scarlet fever reported for January, February, and March 1941, distributed over 3 ships and 9 shore stations. An epidemic of 83 cases occurred at the Naval Training Station, Newport, R. I., in February and March concurrently with an epidemic of German measles. Sporadic cases of scarlet fever had ap-

peared in and around the vicinity of Newport. The origin of the epidemic at the Training Station was problematical. Apparently the focus of infection was among the recruits who had been on the station a month to six weeks and it is possible that contacts were made by recruits while on liberty in Newport.

Despite segregation of infected individuals and contacts; thorough investigation of food handlers, messing facilities, kitchen equipment, and utensils; and investigations to discover a break in hygienic associations which were maintaining the epidemicity of scarlet fever and German measles, there was no appreciable decrease in admissions.

Meningitis, cerebrospinal.—A private, United States Marine Corps, 24 years of age, with 1 month's service, was admitted to the sick list at the Marine Corps Base, San Diego, Calif., on February 4, and died on March 8. The secondary cause of death was endocarditis, subacute, bacterial.

Cerebrospinal fever, meningococcic.—The sanitary report and the communicable disease report from the Marine Detachment, American Embassy, Peiping, China, for the month of March reported the occurrence of one case of cerebrospinal fever, meningococcic. A questionnaire for the case has not been received in the Bureau, consequently no additional information is available.

Diarrhea—Naval Air Station, San Juan, P. R. (Sanitary report for March 1941).—The month of March was characterized by an increase in the number of cases of diarrhea. An effort was made immediately to determine the source, and the following defects were found which directly contributed to this occurrence. The drinking water at the barracks was found to be highly contaminated with bacteria after it had apparently been heated to 180° F. The personnel was advised not to drink tap water. A mechanical defect in the heating facilities and a leakage in the pipes leading to contamination were found and corrected. Ice purchased from a local contractor was found to be highly contaminated. It was immediately recommended that this ice not be used. A semiweekly inspection of drinking fountains and cultures of water and ice was made. The percentage of diarrheas decreased immediately.

U. S. S. Hogan.—(Special report dated June 3, 1941).—On March 15, 1941, 2 cases of diarrhea appeared aboard the U. S. S. *Hogan*. In the next 9 days a total of 26 cases occurred, representing approximately 20 percent of the ship's complement.

All of the cases were characterized by a sudden onset. Diarrhea was the first complaint followed by fever, headache, and chills after the first three or four stools. The average case had 10 to 12 stools per day. The stools were quite watery, and were not malodorous. No blood, mucus, ova, or parasites were found microscopically. The temperatures were 99° F. to 100° F., when seen early, and 104° F. 10

to 12 hours later. The patients responded to treatment within 2 to 3 days regardless of height of fever or number of stools.

The following sources of infection were considered:

1. A shipment of candy received aboard on March 14 and opened for sale on the following day.

2. A shipment of commissary provisions received aboard on March 14.

3. Food products and drinks were available elsewhere, but many persons were sick who had not been ashore or who had not purchased candy or soft drinks from other ships in the nest, and many persons from other ships used these products without becoming ill.

4. The ship's drinking water was used until the second day when water from the tender was used. Since the water aboard ship was turned off from time to time during the day for conservation purposes, several of the crew used water from the after peak tank. Examination of this water microscopically revealed many active flagellated forms and various bacteria.

The following corrective measures were effected:

- (a) The peak tank was padlocked.

- (b) Dish washing was supervised by a pharmacist's mate.

- (c) Mess cooks were examined and cautioned by the Medical Officer.

- (d) The crew was called to quarters and cautioned about the epidemic.

- (e) Tincture of green soap solution was placed in the crew's wash room.

- (f) Candy was withdrawn from sale at the ship's store.

- (g) Living spaces were thoroughly cleaned and woodwork and decks washed with cresol solution.

- (h) Mattresses were sprayed with Navy Standard Insecticide.

Although no definite etiological agent was found and the definite source of the outbreak was not discovered, the Medical Officer believes that the spread of this disease occurred by hand-to-mouth infection, once started.

There have been no new cases of diarrhea aboard this ship since the termination of this outbreak.

Gastrointestinal disorder—U. S. S. Indianapolis—(Special Report dated May 9, 1941).—On February 11, 1941, while moored alongside the U. S. S. *Pennsylvania* at Pearl Harbor, T. H., 5 cases of a gastrointestinal disorder were admitted to the sick list. Cases continued to occur sporadically with a minimum of 4 on the sick list on February 22 to a maximum of 18 on February 28. On March 8 a sharp increase to 36 cases occurred. The condition gradually regressed with sporadic cases until April 24 when the last case was discharged from the sick list. No further cases occurred.

While this vessel was secured to dock all water was obtained from

the Navy Yard, Pearl Harbor. While moored in stream water was received from barge twice during the period March 26 to 29. Fresh water only was used throughout the ship. Repeated analyses of the water from evaporators, tanks, and scuttle-butts were negative. Specimens of milk powder ice cream and ice-cream mix were negative. It is of interest to note that a specimen of milk powder which was mixed with water contained a bacteria count of 4,000 *Coli aerogenes* group per cc., while the water and milk powder before mixing were negative. This would lead to the belief that the original infection was by a carrier, which is further borne out by the fact that all of the cases but one occurred in the general mess. No cases occurred among officers or mess attendants.

The symptoms noted were numerous watery stools within 24 hours, average 12 hours; abdominal cramps; fever 101° F. to 105° F.; tenesmus; and small amounts of blood in stools.

Blood counts were within normal limits.

Stools examined aboard ship showed many pus cells, red blood cells, with a rod-shaped bacilli nonmotile. Culture from cases transferred to the U. S. S. *Relief* showed *Para-dysenteriae* (Hiss-Russell). Stools cultured aboard the U. S. S. *Relief* from cases retained aboard the U. S. S. *Indianapolis* when positive showed the same organism.

All of the cases received an initial purge of paregoric and castor oil, followed at 4-hour intervals by bismuth and paregoric. Lukewarm colonic irrigations of normal saline were routinely used in cases of severe tenesmus. Serum was not available.

The following control measures were instituted:

1. Notices were posted about ship on the method of spread.
2. Paper towels and soap were placed in all heads, advising the use of these after going to toilet.
3. Mess cooks were inspected daily by the medical officer and men who complained of even the slightest symptoms were relieved from duty.
4. Pharmacist's mates were stationed in the scullery.
5. No men admitted with the disease were allowed to return as mess cooks for 30 days.
6. Ship's cooks were not allowed to return to their duty until negative cultures of stools were obtained.
7. All uncooked native fruits and vegetables were eliminated from menu:

The following number of admissions were recorded:

Diagnosis undetermined (bacillary dysentery)-----	3
Diagnosis undetermined (enteritis, acute)-----	1
Bacillary dysentery-----	51
Gastroenteritis, acute -----	76

It is believed that the focus of infection was a carrier in the general mess.

MORBIDITY

Summary for the quarter ending Mar. 31, 1941

TABLE 1.—Total, United States Navy

Average strength.....	Forces afloat		Forces ashore		Entire Navy	
	156, 049		123, 087		279, 136	
	Admissions	Rate per 1,000	Admissions	Rate per 1,000	Admissions	Rate per 1,000
All causes.....	19, 583	501. 97	24, 765	804. 80	44, 348	635. 50
Diseases only.....	17, 864	457. 91	23, 358	759. 07	41, 222	590. 71
Injuries and poisonings.....	1, 719	44. 06	1, 407	45. 72	3, 126	44. 80
Communicable diseases transmissible by oral and nasal discharges (class VIII):						
(A).....	1, 786	45. 78	4, 308	140. 00	6, 094	87. 33
(B).....	6, 342	162. 56	11, 373	369. 59	17, 715	253. 85
Venereal diseases.....	3, 048	78. 13	1, 299	42. 21	4, 347	62. 29

TABLE 2.—Summary of morbidity, naval districts, for the quarter ending Mar. 31, 1941

District	Average strength	Annual rates per 1,000			
		All causes	Diseases only	Respiratory diseases	Venereal diseases
1st Naval District.....	7, 434	1, 500. 67	1, 445. 79	972. 83	24. 21
3d Naval District.....	7, 629	743. 48	692. 10	339. 23	45. 09
4th Naval District.....	4, 143	619. 84	568. 67	349. 50	36. 69
Activities on the Severn and Potomac Rivers.....	13, 733	788. 17	746. 23	436. 61	23. 88
5th Naval District.....	13, 630	1, 075. 57	1, 036. 24	449. 60	58. 40
6th Naval District.....	4, 201	1, 045. 47	983. 58	619. 85	35. 23
7th Naval District.....	5, 492	530. 95	503. 28	306. 63	59. 72
8th Naval District.....	6, 915	669. 27	624. 15	352. 28	37. 60
9th Naval District.....	8, 653	1, 700. 68	1, 662. 31	1, 203. 28	7. 86
10th Naval District.....	2, 637	667. 43	591. 58	128. 93	150. 17
11th Naval District.....	28, 602	494. 37	457. 73	175. 93	28. 67
12th Naval District.....	5, 046	447. 09	406. 66	159. 33	37. 26
13th Naval District.....	3, 868	531. 54	477. 77	174. 77	41. 37
14th Naval District.....	7, 180	321. 45	282. 45	84. 12	21. 17
15th Naval District.....	2, 679	392. 68	337. 44	74. 65	68. 68
16th Naval District.....	1, 724	392. 11	364. 27	32. 48	118. 33
Outlying stations.....	3, 063	985. 96	895. 85	419. 20	156. 71

TABLE 3.—Venereal diseases (class XII) and communicable diseases transmissible by oral and nasal discharges (class VIII), ships and shore stations, for the quarter ending Mar. 31, 1941

	Venereal diseases		Communicable diseases	
	Rate per 1,000, 1st quarter, 1941	Median rate, 1st quarter, 1936-40	Rate per 1,000, 1st quarter, 1941	Median rate, 1st quarter, 1936-40
SHIP				
Albermarle.....	134.92	¹ 12/4/40	246.03	¹ 12/4/40
Altair.....	24.84	64.75	111.80	57.55
Argonne.....	10.94	49.69	235.29	162.29
Arizona.....	55.96	76.41	433.09	297.55
Arkansas.....	84.07	102.33	374.24	232.56
Astoria.....	9.41	58.39	80.00	288.18
Augusta.....	169.18	379.75	302.11	45.57
Barnett.....	207.55	¹ 9/25/40	481.13	¹ 9/25/40
Battle Force, Fleet Air Detachment.....	44.87	41.20	201.91	148.77
Black Hawk.....	414.20	373.63	71.01	77.92
Boise.....	71.57	¹ 8/12/38	318.09	¹ 8/12/38
Brooklyn.....	51.95	¹ 9/30/37	155.84	¹ 9/30/37
California.....	57.61	66.38	425.80	204.91
Canopus.....	332.11	197.22	78.14	96.08
Chaumont.....	216.59	126.32	92.16	184.91
Chester.....	109.13	87.91	310.60	126.38
Chicago.....	14.94	59.95	239.03	197.72
Cincinnati.....	151.82	92.63	33.00	168.00
Colorado.....	58.41	68.35	424.13	96.85
Concord.....	11.30	121.90	112.99	72.46
Curtiss.....	89.07	¹ 11/15/40	178.14	¹ 11/15/40
Denebola.....	142.27	¹ 4/6/40	257.18	¹ 4/6/40
Detroit.....	41.36	48.70	230.43	97.39
Dixie.....	55.75	¹ 4/25/40	144.02	¹ 4/25/40
Dobbin.....	51.50	65.57	97.28	36.14
Enterprise.....	106.46	¹ 5/12/38	471.48	¹ 5/12/38
George F. Elliot.....	67.26	¹ 1/10/41	470.79	¹ 1/10/41
Helena.....	21.05	¹ 9/18/39	189.47	¹ 9/18/39
Henderson.....	112.15	247.60	118.38	119.24
Holland.....	109.12	89.39	391.84	282.35
Honolulu.....	26.14	¹ 6/15/38	100.84	¹ 6/15/38
Houston.....	353.29	113.91	129.54	240.21
Idaho.....	14.88	85.65	190.95	224.11
Indianapolis.....	23.21	74.29	266.92	271.19
Langley.....	375.59	83.33	162.75	212.96
Lexington.....	73.93	70.64	329.67	361.15
Louisville.....	108.23	68.11	500.56	271.90
Marblehead.....	382.55	316.36	93.96	120.09
Maryland.....	35.42	61.16	164.45	169.85
McCawley.....	117.13	¹ 8/6/40	220.05	¹ 8/6/40
Medusa.....	11.28	76.09	118.48	138.53
Melville.....	142.22	63.24	183.70	111.69
Memphis.....	177.47	86.96	252.56	108.70
Milwaukee.....	65.79	126.98	111.84	172.73
Minneapolis.....	17.37	53.55	251.90	157.97
Mississippi.....	12.97	66.39	168.61	232.26
Nashville.....	47.76	¹ 6/6/38	358.21	¹ 6/6/38
Nevada.....	135.37	62.21	278.70	121.84
New Mexico.....	21.51	102.56	95.58	218.38
New Orleans.....	36.91	81.16	235.29	127.54
New York.....	82.42	103.80	79.67	77.85
Northampton.....	41.40	41.54	129.37	125.75
Oklahoma.....	13.30	76.29	316.50	221.62
Omaha.....	62.31	177.94	336.45	220.64
Pennsylvania.....	41.10	98.09	200.91	254.30
Pensacola.....	32.94	60.70	175.58	73.06
Philadelphia.....	15.38	¹ 9/23/37	330.77	¹ 9/23/37
Phoenix.....	12.17	¹ 10/3/38	125.76	¹ 10/3/38
Portland.....	26.32	98.61	65.79	228.04
Prairie.....	80.42	¹ 8/5/40	791.53	¹ 8/5/40
1st Marine Division, F. M. F., Quantico, Va.....	37.41	(²)	370.40	(²)
Quincy.....	144.22	¹ 6/9/36	627.78	¹ 6/9/36
Raleigh.....	36.31	121.00	623.30	347.07
Ranger.....	73.09	87.57	231.88	400.00
Relief (duty personnel).....	62.28	70.89	193.77	212.66
Richmond.....	166.17	66.05	160.24	54.90
Salt Lake City.....	131.15	77.42	505.85	71.86
San Francisco.....	34.86	85.91	139.43	237.87
Saratoga.....	93.30	66.07	559.96	225.23
Savannah.....	78.43	¹ 3/10/38	251.81	¹ 3/10/38
St. Louis.....	53.89	¹ 5/19/39	49.74	¹ 5/19/39
Tennessee.....	34.23	93.62	254.28	103.16
Texas.....	95.98	108.21	285.11	133.44
Trenton.....	100.00	130.54	93.75	323.58

¹ Commissioned.² Not available.

TABLE 3.—Venereal diseases (class XII) and communicable diseases transmissible by oral and nasal discharges (class VIII), ships and shore stations, for the quarter ending Mar. 31, 1941—Continued

	Venereal diseases		Communicable diseases	
	Rate per 1,000, 1st quarter, 1941	Median rate, 1st quarter, 1936-40	Rate per 1,000, 1st quarter, 1941	Median rate, 1st quarter, 1936-40
SHIP—continued				
Tuscaloosa.....	262.22	97.56	40.00	222.22
Utah.....	89.80	79.60	220.41	402.28
Vestal.....	82.54	30.82	317.46	170.65
Vincennes.....	159.14	¹ 2/24/37	262.37	¹ 2/24/37
Wasp.....	202.82	¹ 4/25/40	110.40	¹ 4/25/40
West Virginia.....	53.27	53.93	273.31	220.75
Whitney.....	30.67	67.32	73.62	130.51
Wichita.....	99.48	¹ 2/16/39	277.72	¹ 2/16/39
Wyoming.....	66.83	114.81	133.67	160.73
Yorktown.....	20.55	¹ 9/30/37	121.00	¹ 9/30/37
STATION				
Naval Air Station, Anacostia, D. C.	27.68	10.34	525.95	90.45
Naval Academy (midshipmen), Annapolis, Md.	0	1.78	527.88	625.11
Naval Academy (other than midshipmen), Annapolis, Md.	82.44	50.11	439.69	297.16
Marine Barracks, Quantico, Va.	27.54	37.17	656.22	161.01
Marine Barracks, Washington, D. C.	40.61	0	1,025.38	394.56
Naval Dispensary, Washington, D. C.	0	(²)	621.25	(²)
Navy Yard, Washington, D. C.	25.68	34.40	196.90	167.46
Navy Yard, Boston, Mass.	17.19	19.85	429.80	195.37
Navy Yard, Portsmouth, N. H.	21.94	21.51	277.88	290.32
Naval Torpedo Station, Newport, R. I.	44.16	25.10	347.00	139.65
Naval Training Station, Newport, R. I.	6.62	2.87	1,767.24	1,063.47
Submarine Base, New London, Conn.	36.62	38.30	322.58	234.82
U. S. N. R. Radio School, Noroton, Conn.	0	(²)	728.52	(²)
Naval Hospital, Brooklyn, N. Y.	96.87	(²)	478.63	(²)
District Headquarters, New York, N. Y.	13.58	11.70	529.71	80.00
U. S. N. R. Midshipmen's School, New York, N. Y.	24.62	¹ 8/1/40	24.62	¹ 8/1/40
Navy Yard, New York, N. Y.	25.36	62.50	285.26	146.21
Receiving Ship, New York, N. Y.	71.02	97.22	471.94	330.60
Naval Air Station, Lakehurst, N. J.	28.78	35.61	294.96	271.70
Naval Hospital, Philadelphia, Pa.	9.20	(²)	496.55	(²)
Navy Yard, Philadelphia, Pa.	42.47	24.69	403.45	249.67
Receiving Station, Philadelphia, Pa.	63.22	65.57	227.61	172.76
Naval Air Station, Norfolk, Va.	75.45	70.33	418.38	193.41
Receiving Station, Norfolk, Va.	176.10	99.75	352.20	160.40
Naval Training Station, Norfolk, Va.	40.12	45.19	763.49	1,009.22
Norfolk Naval Hospital, Portsmouth, Va.	36.20	(²)	841.63	(²)
Norfolk Navy Yard, Portsmouth, Va.	47.69	72.46	681.63	319.65
Navy Yard, Charleston, S. C.	67.92	15.38	762.26	170.39
5th Defense Battalion, F. M. F., Parris Island, S. C.	20.49	¹ 12/1/40	1,024.33	¹ 12/1/40
Marine Barracks, Parris Island, S. C.	31.55	(²)	519.72	(²)
Naval Air Station, Jacksonville, Fla.	91.82	¹ 10/15/40	568.67	¹ 10/15/40
Naval Station, Key West, Fla.	28.79	¹ 11/1/39	88.59	¹ 11/1/39
Naval Air Station, Key West, Fla.	48.14	(²)	293.22	(²)
Naval Air Station, Pensacola, Fla.	37.87	41.71	468.16	264.15
Naval Air Station, Corpus Christi, Tex.	60.58	¹ 3/12/41	189.30	¹ 3/12/41
U. S. N. R. Midshipmen's School, NW. U., Chicago, Ill.	0	¹ 9/16/40	896.80	¹ 9/16/40
U. S. N. R. Signal School, Chicago, Ill.	11.05	¹ 10/—/40	1,414.35	¹ 10/—/40
Naval Hospital, Great Lakes, Ill.	0	(²)	1,155.08	(²)
Naval Training Station, Great Lakes, Ill.	9.65	5.08	1,564.30	483.97
4th Defense Battalion, F. M. F., Guantanamo Bay, Cuba	143.59	¹ 2/1/40	1,035.90	¹ 2/1/40
Mobile Base Hospital No. 1, Guantanamo Bay, Cuba	149.25	¹ 10/5/40	189.05	¹ 10/5/40
Naval Station, Guantanamo Bay, Cuba	183.21	88.33	178.84	49.04
Naval Air Station, San Juan, P. R.	119.34	¹ 9/18/39	53.04	¹ 9/18/39
Dispensary, 11th Naval District, San Diego, Calif.	0	0	248.63	15.83
Marine Corps Base, San Diego, Calif.	36.70	30.70	281.66	138.76
Naval Air Station, San Diego, Calif.	30.96	21.64	379.63	89.47
Naval Hospital, San Diego, Calif.	23.90	(²)	653.39	(²)
Receiving Station, San Diego, Calif.	53.56	48.25	199.78	40.76
Naval Training Station, San Diego, Calif.	8.00	9.76	274.95	291.80
Naval Air Station, San Pedro, Calif.	27.10	¹ 7/1/37	102.98	¹ 7/1/37
Navy Yard, Mare Island, Calif.	24.96	29.02	305.77	92.59
District Headquarters, San Francisco, Calif.	0	(²)	223.46	(²)
Receiving Ship, San Francisco, Calif.	89.33	111.32	158.81	103.90
Puget Sound Navy Yard, Bremerton, Wash.	55.11	34.72	304.57	98.77
Naval Air Station, Seattle, Wash.	43.66	48.19	241.81	195.12

¹ Commissioned.

² Not available.

TABLE 3.—Venereal diseases (class XII) and communicable diseases transmissible by oral and nasal discharges (class VIII), ships and shore stations, for the quarter ending Mar. 31, 1941—Continued

	Venereal diseases		Communicable diseases	
	Rate per 1,000, 1st quarter, 1941	Median rate, 1st quarter, 1936-40	Rate per 1,000, 1st quarter, 1941	Median rate, 1st quarter, 1936-40
STATION—continued				
2nd Marine Air Group, F. M. F., Ewa, Oahu, T. H.	0	(²)	111.80	(²)
Naval Air Station, Kaneohe Bay, Oahu, T. H.	0	¹ 2/15/41	92.70	¹ 2/15/41
1st Defense Battalion, F. M. F., Pearl Harbor, T. H.	39.06	(²)	109.36	(²)
3d Defense Battalion, F. M. F., Pearl Harbor, T. H.	15.54	(²)	202.07	(²)
Naval Air Station, Pearl Harbor, T. H.	25.18	24.84	83.50	49.69
Navy Yard, Pearl Harbor, T. H.	14.60	22.04	62.04	44.08
Submarine Base, Pearl Harbor, T. H.	16.13	29.78	120.97	107.32
Naval Air Station, Coco Solo, C. Z.	17.13	60.02	45.68	73.82
Submarine Base, Coco Solo, C. Z.	165.44	124.51	159.53	141.37
Navy Yard, Cavite, P. I.	132.90	168.28	19.45	54.47
Marine Detachment, Peiping, China	262.30	163.44	491.80	421.51
4th Marines, Shanghai, China	216.12	195.46	265.71	339.89
Marine Detachment, Tientsin, China	247.42	(²)	371.13	(²)
Marine Barracks, Guam, Marianas Islands	130.72	55.56	52.29	35.40
Naval Hospital, Guam, Marianas Islands	46.51	(²)	46.51	(²)
Naval Station, Guam, Marianas Islands	17.86	36.87	89.29	47.24
7th Defense Battalion, F. M. F., Tutuila, Samoa	0	(²)	364.32	(²)
Naval Station, Tutuila, Samoa	130.43	25.16	195.65	75.47

¹ Commissioned.² Not available.**DEATHS**

During the quarter ending Mar. 31, 1941

Cause		Total	Navy		Marine corps		Nurse Corps	Midshipmen
Primary	Secondary		Officers	Men	Officers	Men		
Average strength		279, 136	18, 422	208, 472	2, 786	46, 638	545	2, 273
<i>Diseases</i>								
Abscess, thigh	Septicemia	1		1				
Abscess, mediastinum		1		1				
Abscess, peritonsillar	Embolism, pulmonary	1		1				
Aneurysm, aorta	Atelectasis	1		1				
Appendicitis, acute	Peritonitis, general, acute	2		1		1		
Do	Thrombosis, pulmonary artery.	2	1			1		
Atrophy, liver, yellow, acute.		1		1				
Blackwater fever	Endocarditis, acute, ulcerative.	1		1				
Carcinoma, histologic type unspecified, mesentery.		1				1		
Carcinoma, histologic type unspecified, sigmoid colon.		1		1				
Carcinoma, histologic type unspecified, stomach.	Peritonitis, general, acute	1		1				
Coronary heart disease, arteriosclerotic.		2	1	1				
Dementia praecox		1		1				
Dilatation, cardiac, acute		2		1		1		
Encephalitis, acute		2		2				
Encephalitis, lethargic		1		1				
Epilepsy		1		1				
Filariasis, abscesses, multiple.	Toxemia, bacterial	1		1				
Hemorrhage, cerebral		1		1				
Hernia, inguinal, indirect	Poisoning, anesthesia, procaine.	1		1				

DEATHS—Continued
During the quarter ending Mar. 31, 1941

Cause		Total	Navy		Marine corps		Nurse Corps	Midshipmen
Primary	Secondary		Officers	Men	Officers	Men		
<i>Diseases—Con.</i>								
Hypertensive heart disease.	Thrombosis, cerebral	1	1					
Infarction, brain		1		1				
Influenza	Pneumonia, lobar	1		1				
Lymphosarcoma, stomach		1		1				
Malaria, malignant tertian		1				1		
Meningitis, cerebrospinal, acute.	Endocarditis, sub-acute, bacterial.	1				1		
Obstruction, intestinal, from external causes.		1		1				
Otitis media, chronic	Mastoiditis, acute	1		1				
Peritonitis, general, acute	Thrombosis, right colic artery.	1				1		
Pneumonia, broncho.		1		1				
Do	Pneumonitis, chronic, non-tuberculous.	1		1				
Do	Tracheobronchitis, acute	1		1				
Pneumonia, lobar	Dilatation, cardiac, acute	1		1				
Rheumatic fever	Pericarditis, acute	1		1				
Sarcoma, tibia		1		1				
Thrombosis, coronary artery.		13	2	8	3			
Tuberculosis, pulmonary, acute general miliary.		1		1				
Total for diseases		54	5	39	3	7	0	0
<i>Injuries and poisonings</i>								
Burn, multiple		6	1	1		4		
Crush, chest		1		1				
Crush, head		2				2		
Crush, vertebrae, cervical		1			1			
Drowning		14	1	9		4		
Drowning	Intracranial injury	1	1					
Fracture, comp. skull		9	1	5		3		
Fracture, comp. skull	Psychosis, unclassified	1			1			
Fracture, simple, ribs	Wound, punctured, liver and lung.	1		1				
Fracture, simple, ribs	Wound, punctured, lung.	1		1				
Fracture, simple, skull	Hemorrhage, traumatic, subdural.	1		1				
Fracture, simple, vertebrae, cervical.	Intraspinal injury	1	1					
Fracture, simple, vertebrae, cervical.	Myelitis, transverse	1		1				
Injuries, multiple, extreme.		32	10	22				
Injuries, multiple, extreme.	Psychosis, unclassified	1				1		
Intracranial injury		3		1		2		
Strangulation, respiratory	Psychosis, unclassified	1		1				
Wound, gunshot, head		6	1	4		1		
Wound, gunshot, head	Melancholia, involutional.	1	1					
Wound, gunshot, head	Psychoneurosis, anxiety neurosis.	1		1				
Wound, gunshot, head	Psychosis, unclassified	1		1				
Wound, punctured, abdomen.	Abscess, lung	1		1				
Wound, punctured, chest.	Peritonitis, general, acute.	1		1				
Wound, punctured, heart.		1		1				
Poisoning, acute:								
Barbiturates		1	1					
Cyanide		1	1					
Gasoline fumes		1		1				
Illuminating gas		1		1				
Poisoning, therapeutic, acute, gonococcus infection, urethra.	Granulocytopenia, malignant.	1		1				
Total for injuries and poisonings.		94	19	56	2	17	0	0
Grand total		148	24	95	5	24	0	0
Annual death rate per 1,000:								
All causes		2.12	5.21	1.82	7.18	2.06		
Diseases only		.77	1.09	.75	4.31	.60		
Drowning		.21	.43	.17		.34		
Poisoning		.07	.43	.06				
Other injuries		1.06	3.26	.84	2.87			

MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

Statistics for the quarter ending Mar. 31, 1941

The following statistics were taken from sanitary reports submitted by naval training stations.

January, February, and March	United States naval training station—			
	Norfolk, Va.	Newport, R. I.	Great Lakes, Ill.	San Diego, Calif.
Recruits received during the period.....	6, 752	3, 737	7, 414	5, 951
Recruits appearing before Board of Medical Survey.....	91	70	1	49
Recruits recommended for discharge from the service.....	91	60	17	49
Recruits discharged by reason of medical survey.....	53	(1)	6	(1)
Recruits held over pending further observation.....	76	10	(1)	0
Recruits transferred to the hospital for treatment, operation, or further observation for conditions existing prior to enlistment.....	(1)	116	(1)	90

¹ Not reported.

The following table was prepared from reports of medical surveys in which disabilities or disease causing the survey were noted existing prior to enlistment. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office.

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Absence, acquired, teeth.....	15	Constitutional psychopathic state, inadequate personality.....	21
Albuminuria.....	1	Constitutional psychopathic state, paranoid personality.....	1
Allergy.....	1	Constitutional psychopathic state, sexual psychopathy.....	4
Amblyopia.....	1	Cryptorchidism.....	1
Appendicitis, chronic.....	2	Curvature, spine.....	2
Arthritis, chronic.....	6	Cyst.....	1
Asthma.....	6	Cystitis, chronic, nonvenereal.....	1
Astigmatism, compound, myopic.....	2	Deafness, bilateral.....	9
Atrophy.....	4	Deafness, unilateral.....	4
Cardiac arrhythmia, premature contractions.....	1	Defective physical development.....	4
Cardiac arrhythmia, paroxysmal tachycardia.....	1	Deformity, acquired.....	7
Caries, teeth.....	7	Deformity, congenital.....	8
Cataract.....	1	Dementia praecox.....	9
Choroiditis.....	1	Dislocation, articular cartilage.....	3
Cicatrix, skin.....	2	Dislocation, chronic.....	5
Color blindness.....	3	Effort syndrome.....	3
Constitutional psychopathic inferiority, without psychosis.....	15	Encephalitis, chronic.....	1
Constitutional psychopathic state, emotional instability.....	23	Endocrinopathy (pituitary).....	1
		Enlarged inguinal rings.....	1

Cause of survey	Num-ber of sur-veys	Cause of survey	Num-ber of sur-veys
Enuresis	49	Psoriasis	1
Epilepsy	18	Psychoneurosis, anxiety neurosis	9
Epiphyseal separation, traumatic	1	Psychoneurosis, compulsion neurosis	1
Fibroma, tendo-achilles	1	Psychoneurosis, situational	1
Flat foot	28	Psychoneurosis, hysteria	12
Foreign body, traumatic	1	Psychoneurosis, neurasthenia	3
Fracture, vertebra, compression	1	Psychoneurosis, psychasthenia	1
Gastritis, acute	1	Psychoneurosis, traumatic	7
Glycosuria	1	Psychoneurosis, unclassified	3
Goiter, simple	2	Ptosis	1
Gonococcus infection, prostate	1	Rheumatic fever, acute	4
Hammer toe	1	Sclerosis, disseminated	1
Hay fever	1	Sinusitis, frontal, chronic	2
Hematuria	1	Situs inversus viscerum	1
Hernia, inguinal, indirect	13	Somnambulism	13
Hernia, inguinal, direct	2	Spondylolisthesis	1
Hernia, ventral	1	Spondylitis	1
Hydrocele	2	Sprain, joint, chronic	3
Hydrocele, tunica vaginalis	3	Strain, muscle	1
Hypertension, arterial	5	Syncope	1
Hypochondriasis	1	Syphilis	2
Hypoglycemia	1	Syphilis, sero-positive only	1
Ichthyosis	1	Tachycardia	1
Joint, internal derangement	4	Thrombosis, veins	1
Malocclusion, teeth	2	Tic	1
Mastoiditis, chronic	1	Trachoma	1
Migraine	4	Tuberculosis, pulmonary, chronic, active, moderately advanced	9
Myopia	11	Tuberculosis, pulmonary, chronic, arrested, incipient	3
Myositis, chronic	3	Ulcer, duodenum	10
Narcolepsy	1	Ulcer, stomach	1
Nephritis, chronic	11	Union of fracture, faulty	1
Neuralgia	1	Valvular heart disease, aortic insufficiency	2
Neurosis, cardiac	1	Valvular heart disease, mitral insufficiency	5
Nostalgia	1	Valvular heart disease, mitral stenosis	4
Osgood-Schlatter disease	1	Varicocele	1
Osteochondritis, deformans	1	Varicose, veins	2
Osteochondritis, dissecans	1		
Osteochondroma	1		
Osteoma	2		
Osteomyelitis	2		
Otitis media, chronic	28		
Pansinusitis	1		
Paradentosis	1		
Pes cavus	4		
Pompholyx	1		
		Total	499

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